DISEASES OF THE HEART

BOOKS BY SIR THOMAS LEWIS

CLINICAL DISORDERS OF THE HEART BEAT

CLINICAL ELECTROCARDIOGRAPHY

THE MECHANISM AND GRAPHIC REGISTRATION OF THE HEART BEAT

THE BLOOD VESSELS OF THE HUMAN SKIN AND THEIR RESPONSES

CLINICAL SCIENCE ILLUSTRATED BY PERSONAL EXPERIENCES

VASCULAR DISORDERS OF THE LIMBS DESCRIBED FOR STUDENTS AND PRACTITIONERS

THE SOLDIER'S HEART AND THE EFFORT SYNDROME

PAIN

EXERCISES IN HUMAN PHYSIOLOGY (PRELIMINARY TO CLINICAL WORK)

DEPARTMENT OF CLINICAL RESEARCH,
UNIVERSITY COLLEGE HOSPITAL, LONDON

DISEASES OF THE HEART

DESCRIBED FOR PRACTITIONERS AND STUDENTS

By SIR THOMAS LEWIS
C.B.E., F.R.S., M.D., D.Sc., LL.D., F.R.C.P.
PHYSICIAN IN CHARGE OF DEPARTMENT OF CLINICAL RESEARCH, UNIVERSITY COLLEGE HOSPITAL LONDON, HONORARY CONSULTING PHYSICIAN TO THE MINISTRY OF PENSIONS; CONSULTING PHYSICIAN, CITY OF LONDON HOSPITAL, FELLOW OF UNIVERSITY COLLEGE, LONDON

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PREFACE TO FOURTH EDITION

If I had set out to describe the many and often very complex methods of examination now in use, had attempted to discuss at any length the pathology of heart disease, had included all known disorders of the heart, giving to each of these the space commensurate with our knowledge of it, and without consideration of its commonness or rarity, this book would have grown to form an unwieldy treatise The impulse to write a work of reference has not stirred me, but I have had the desire to place at the disposal of students and medical practitioners the outline of my clinical teaching on diseases of the heart, as this has developed in my talks to my own hospital students

In beginning that teaching thirty and more years ago, I determined that the basis of what I taught should be that which I myself had seen and proved to be true A second ideal that I have striven hard to attain is simplicity in teaching, this was not so easy during the years in which my work lay largely among pulse and galvanometric curves But I have become more and more convinced of the need for simplicity and it was apparent that little of the graphic work could profitably be taught to men entering practice, whose foremost interest must always be in observations they themselves can make upon their patients

Such a vast amount of knowledge has accumulated about human disease, that we can now cut ruthlessly away from text books all that we perceive to rest upon an insecure basis, and we can dispense with many trivial details having something more substantial to put in their places Some space has been regretfully given to the explanation of such pruning Otherwise I have endeavoured to keep to what practice needs and to discuss such things thoroughly I have tried to strip my subject of intricacies and redundances, of unnecessary technical terms, named signs, and the old trite phrases, for these begin to stifle Medicine It has been important to try to achieve a proper perspective of values so as not to place undue weight on this or that, because its novelty attracts or because it has a strong personal interest I shall not be accused, I think, of over-emphasising electrocardiography The few graphic records published suffice only to show the chief directions in which these records prove serviceable Radiography I have treated similarly, by using illustrative orthodiagrams, chosen from a large collection of

my own taking In the book, chief emphasis has fallen upon physical signs that are within the immediate reach of all I have laid deliberate stress on common forms of disease, but here and there use the rare when it illuminates

What is said I have tried to arrange whenever possible in a natural and logical sequence of ideas and not simply as a series of discon nected facts There is abundant opportunity in the study of patients for the exercise of thought, and no system of teaching that relies chiefly upon memory, or neglects to foster processes of reason ing can have real educational merit A chief reform needed in medical education to day is that students should be encouraged to take a more leisurely and intelligent interest in phenomena, not of the laboratory, with which they will soon lose all connection, but of everyday practice An underlying philosophy, when it can be found is invaluable in practice, not only because it quickens and maintains interest but because it forms a stable guide to action when experience fails as it often will in face of the unusual, to give precise or particular direction I have tried and perhaps not always with success to follow and obey the recognised law that there is but one cause for one effect and to group symptoms where they belong, so that the same group is not accredited to two distinct lesions, but to one common state

And now to consider the arrangement of the subject matter It is traditional to classify diseases whenever possible upon an anatomi cal basis My first reaction to an anatomical classification of heart disease is that heart diseases cannot be classified Medicine is top heavy with classifications which often illogical parody those of the systematic biologies My second reaction is that anatomy fails as a chief basis of thought where heart disease is concerned When Surgery classifies and likewise thinks anatomically, it has at least the excuse that it is so impelled because anatomy rigidly governs treatment of the diseases falling within its sphere Medicine has the privilege of greater freedom, can view the body more as one whole, see disease more broadly, and picture a series of states of disordered function recognisable only during life Models of heart disease cannot be cast in anatomical moulds and the persevering efforts so to fashion them have provided, and continue still to provide in our text books images of disease that are often so grotesque in their representation that they largely fail in their excellent purpose of forming guides to the management of disease This is said in deliberately vigorous terms, that it may draw and hold attention It is with the symptoms

of disease that the patient, and that the doctor mainly, contends, and the symptoms of heart disease may be said to derive from faults in function Therefore, in managing our patients, our thoughts must be chiefly set in terms of function and not of structure To whom I fail to teach this first simple, but essential lesson I have nought to teach Who consents to it provisionally, or who grasps it, will be encouraged to pursue the idea by this book's arrangement For it is with this object in mind that I have departed from past precedents and have placed in the foreground those things an understanding of which is of supreme importance in the management of heart cases, namely, cardiac failure and angina pectoris The book differs much in its arrangement and in its outlook from other works dealing with heart disease, but that is perhaps the soundest reason for its publication

The text of the last edition has been revised thoroughly, and a number of changes have been made in the book to keep it in close conformity with recent new work and thought The main changes are in the chapters that deal with cardiac failure

My grateful thanks are again due to my secretary Miss L M Searle who has given me considerable help in the preparation of this edition.

THOMAS LEWIS

CONTENTS

CHAPTER I

CARDIAC FAILURE BREATHLESSNESS

CHAPTER II

SYSTEMIC VENOUS CONGESTION

CHAPTER III

PULMONARY CONGESTION OEDEMA AND INFARCTION

CHAPTER IV

CARDIAC OEDEMA

CHAPTER V

FAILURE WITH CONGESTION (CAUSE, TYPES, PROGNOSIS, AND TREATMENT)

CHAPTER VI

SKIN COLOUR AND CIRCULATORY RATE

CHAPTER VII

CARDIAC ISCHAEMIA CORONARY OCCLUSION

CHAPTER VIII

ANGINA PECTORIS

CHAPTER IX

MINOR PULSE IRREGULARITIES

CHAPTER X

TACHYCARDIAS

CHAPTER XI

AURICULAR FIBRILLATION (IRREGULAR TACHYCARDIA)

CHAPTER XIV

DISEASE OF THE AORTIC VALVE

CHAPTER XV

DISEASE OF THE AURICULO VENTRICULAR VALVES

CHAPTER XX

BACTERIAL ENDOCARDITIS

CHAPTER XXI

RHEUMATIC CARDITIS

CHAPTER XXII

CHRONIC RHEUMATIC HEART DISEASE

CHAPTER XXIII

SYPHILIS OF HEART AND AORTA

CHAPTER XXIV

ESSENTIAL HYPERTENSION

CHAPTER XXV

ARTERIOSCLEROSIS SENILE HEART MYOCARDIUM

CHAPTER XXVI

PULMONARY HYPERTENSION COR PULMONALE FAILURE IN PULMONARY DISEASE RIGHT SIDED FAILURE

CHAPTER XXVII

THYROTOXIC STATE

CHAPTER XXVIII

CONGENITAL MALFORMATION

CHAPTER XXIX

CHILD-BEARING. ANAESTHETICS AND OPERATIONS

CHAPTER XXX

DIAGNOSIS, PROGNOSIS, AND TREATMENT

CHAPTER I

CARDIAC FAILURE BREATHLESSNESS

The Central Problem

It is impossible thoughtfully to survey in the light of daily ex perience the field of medical work covering diseases of the heart varied as the manifestations may be without realising the central problem to be failure of the heart to accomplish its function in lesser or greater degree This function consists in the efficient propulsion of fully aerated blood through the circle of vessels in adequate quantity to meet the needs of the body in the ordinary and varied circumstances of life The very essence of cardiovascular practice is recognition of early heart failure and discrimination between its different grades This simple truth is not stated here for the first time in theory it receives occasional homage from many It emerges into view for a fleeting moment to retreat and lie concealed beneath a mass of technical and by comparison trivial detail it does not dominate cardiac practice as it should When a patient seeks advice and heart disease is suspected or is known to be present two questions are of chief importance Firstly has the heart the capacity to do the work demanded of it when the body is at rest? Secondly what is its capacity in reserve? These questions can be answered broadly and correctly in almost all cases by simple interrogations and by bedside signs and the answers force all other considerations into the background in most cases of chronic heart disease they are essentials to sound prognosis and treatment

Cardiac Failure (General Statement)

Cardiac failure comprises complex problems I shall try to make the approach simple by at first examining failure along the broad lines of its failure as a whole And this is sound because it is the way in which the heart usually presents failure

Cardiac failure signifies inability of the heart adequately to discharge the blood brought to it and such failure culminates in

pooling of blood behind the driving chambers, in the manifestations of the familiar systemic, and of pulmonary, venous congestion Now in the progress of failure these phenomena are late, and they mean that the heart has failed in such degree that it has no longer the capacity to do the work required of it while the body is at rest In much earlier phases of cardiac failure, before signs of congestion have appeared, the underlying defect shows itself in a lack of reserve by means of the cardinal symptom breathlessness on effort In this book I shall use the words "cardiac failure" consistently to mean failure at any stage of its development, when I wish to indicate the last stages, I shall speak of "failure with congestion", when desirable differentiating between systemic and pulmonary congestion

We may often trace the development of the malady as a whole, and correlate its several phases by considering the events as they succeed each other when heart failure comes insidiously and gradually, the reserves being first weakened and the defect displaying itself transiently, the reserves becoming more reduced, and ultimately lost, when the incapacity of the heart is paraded constantly Breathlessness is not in itself abnormal All people, however healthy, become breathless on exercise The abnormality consists in the occurrence of breathlessness in unusual circumstances The first

same breathlessness continues to be experienced, but it is experienced in response to an amount of work that decreases as time passes This progression may be gradual and spread over months or years, or it may occur in definite steps Cardiac breathlessness is to be graded not by the degree of distress it occasions, but by the amount of physical effort that provokes it Breathlessness occurs on walking quickly or uphill, on walking on the flat, on walking slowly short distances later it is present at rest, these are the grades It is in the last two stages that the early signs of actual venous conges

...athlessness at rest be
...plete course
...athlessness

ranging in its degree from normal breathlessness on active exertion to constant distress when the body rests completely

When the heart fails to eject the blood brought to it at rest that blood collects on the venous side and the patient begins to manifest

congested systemic veins, enlarged liver, cyanosis, a high coloured scanty urine, ascites, dropsy of the legs or widespread oedema (anasarca), and in lesser, greater, or predominant degree congestion and oedema of the lungs

Thus cardiac failure may be divided into two stages the stage of symptoms or waning reserve, and the stage of signs or of circulatory embarrassment and breakdown It is a matter of great importance that the full extent of the normal heart's reserve power should be appreciated What the heart is called upon to do under resting conditions is but a small fraction of that of which it is capable If the total capacity of the healthy heart for work is taken at ten units, it is a fairly accurate estimate to say that one unit suffices to maintain a normal circulation while the body is at rest, and that the remaining nine form the reserve Consequently, in considering the development of failure, it is to be realised that by the time congestion sets in, nine tenths of the heart's capacity to perform its tasks have been lost Congestion is not an early but a late manifestation of failure, breathlessness on effort is the early one

Breathlessness Confined to Effort

The first indication of cardiac failure is nearly always to be found in a diminished tolerance of exercise Of the very numerous tests of cardiac efficiency and inefficiency that have been devised, based as they are mainly upon pulse rate or upon blood pressure or upon both, there is none that approaches in delicacy the symptom breathlessness For this reason, and because undue breathlessness on effort is the commonest complaint of cardiac patients, it is the symptom that takes first place in our work

The most important gauge of a reduced respiratory reserve is to be obtained, when dealing with an intelligent patient, from himself He tells us that some act previously undertaken without noticeable distress, has recently begun to cause breathlessness or undue breathlessness It is generally redundant to set up specific exercise tests against such a gauge as this No test that we can devise however carefully graded it may be will impose the same strain upon or will *yield the same response in, different individuals unaccustomed to it* In gauging finally, a comparison between a healthy and an unhealthy individual is usually far less satisfactory than between the healthy and unhealthy states of the same subject In estimating the ease with which breathlessness is called forth, a careful interrogation of

the patient is to be placed first, not only for the reason given, but for another that is equally important For this interrogation, while acquainting us with the patient's bodily activities in his daily life, in work and in play, brings us at once into contact with the precise difficulties which the patient experiences, and it forms the source of just the information that will be required when the control of bodily work for purposes of treatment comes to be considered The questioning should be close, therefore, and should cover a number of activities, so that, while full information is obtained, there may be an opportunity of observing consistency or inconsistency But all patients are not intelligent, neither are all accurate in their statements, and grave inaccuracies are especially liable to appear when the examination is conducted for a public service or for purposes of insurance It is desirable, therefore, to be familiar with normal reactions to simple forms of test exercise It is undesirable that tests should become too stereotyped, or that they should be regarded as exact measures, a test that is convenient in one circumstance is inconvenient in another It is important that the tests employed should be few in number, so that the reactions to them may be the easier to know and that the exercise performed should be simple and natural No more than one or two tests are required in any given case It is a rule that no patient who exhibits breathlessness when standing still after undressing or while lying at rest, should be tested The tests in these are indeed wholly unnecessary, since it is already clear that there is no reserve If this lack of reserve is cardiac in origin the early or later signs of venous congestion will nearly always be found

SIMPLE TEST

The subject walks briskly up a flight of forty steps, one step at a time and is examined at once As an equivalent, twenty hops on

tests is so satisfactory as, though often more convenient than stairs, since they are less accustomed acts A young subject in good health, but leading a sedentary life will show in this test little respiratory reaction, breathing will show no obvious hurry or material increase of depth, questions will be answered without interruption by respiratory movement The pulse will rise no more than 10 to 20

beats per minute, and the original rate will be resumed within a minute or a little more

A number of cardiac subjects will show manifestly disturbed respiration on this test They are subjects who usually complain voluntarily of breathlessness on walking upstairs or briskly on the flat, and belong, when the breathlessness is of cardiac origin, to a class in which the reserves are largely exhausted, but not as yet to the point of circulatory breakdown The simple test can be employed with safety in the case of any patient who has walked to the consultation, or who shows no breathlessness on undressing

STRENUOUS TEST

To produce breathlessness is the object of this test A weight of ten to twenty pounds is raised from the floor, the arms flexing easily and then extending, above the head at the rate of one lift in two seconds A healthy young man of sedentary habit can accomplish this lift thirty to sixty times, but at the end he will be so breathless [illegible]

[illegible] exercise should be stopped if, during the exercise, the mouth opens in inspiration, or if there is any other clear sign of respiratory embarrassment The number of the lifts is the gauge of the reserve. This test is only to be employed upon subjects who have passed the simple test, its effects should be watched closely while it is in progress, and the exercise stopped as soon as breathlessness is obvious In these circumstances it can be employed safely, irrespective of what has been found in the heart

Cardiac cases may be divided, from the standpoint of heart failure, somewhat arbitrarily into four classes

(1) *Good exercise tolerance* —Able to engage in all but the more strenuous forms of exercise and work, passing the strenuous test

(2) *Fair exercise tolerance* —Able to walk on the flat, at a good pace, and upstairs without stopping or distress, become breathless on hurrying and on hills, fail to pass the strenuous test

(3) *Poor exercise tolerance* —Complain of breathlessness on walking upstairs or briskly on the flat Fail to pass the simple test

(4) *No exercise tolerance* —Breathless when lying at rest or on the slightest exertion These patients usually display signs of venous congestion in greater or lesser degree

Breathlessness at Rest

The term dyspnoea means breathlessness or difficult breathing Hyperpnoea is a distinct term and is equivalent to increased ventilation

It has long been recognised in examining the relation of breath lessness at rest to other phenomena of cardiac failure, that its association with pulmonary engorgement seems closest The latter has very naturally come to be regarded as a main cause of the conspicuous breathlessness of cardiac failure in resting patients For engorgement of the minute vessels diminishes the alveolar space and in individual alveoli abolishes it Engorgement increases transudation, and this fluid, appearing in the alveoli or small bronchi also aids in decreasing their spaces The air spaces of the lungs may be further encroached upon by enlargement of the heart, by pleural effusion and by a diaphragm raised upon an enlarged liver or ascitis There results, in greater or less degree, a reduction of vital capacity and a relative increase in the dead space The tissues of the congested lungs are less resilient than normal and the effort to inflate and deflate them is increased A given tidal flow requires an abnormal effort and this tidal flow gives but a reduced inflow to the alveoli Thus ventilation while rendered difficult is also impaired With ventilation impaired aeration of blood in its passage through the lungs suffers a defect of gaseous interchange which thickening of the alveolar wall tends to exaggerate

An increased ventilation is demanded to maintain normal or even subnormal blood aeration The manner in which hyperpnoea comes has been a matter of prolonged investigation and discussion the factors inducing dyspnoea and bringing compensatory hyperpnoea are undoubtedly complex and final and particular statements cannot always be made Several factors are known or thought to contribute If blood leaves the heart and travels to the centres of respiratory regulation (sinus caroticus and brain) in a state of deficient aeration it will stimulate these centres If blood travels to these centres in inadequate quantity the effect will be similar Congestion of the lung itself directly stirs respiratory reflexes quickening respiration encroachment upon the air spaces by pressure from without acts similarly These pulmonary reflexes are to day regarded as the chief source of hyperpnoea in those who become breathless at rest, a defective output of oxygenated blood is regarded as a chief con tributory factor Lastly, there are indications that on occasion the

blood leaving the heart may be more acid than normal, because it has retained an unusual amount of CO_2, or because its content of non volatile acid is raised, the first occurs only in advanced congestion, the latter occurs in some hypertensive cases and with suspected or actual renal defects

This is the broad outline of present views, but they remain incomplete They refer particularly to hyperpnoea that is present at rest and is exaggerated by exercise, and to breathlessness that appears at rest Breathlessness that comes only during exercise is less clearly understood

Orthopnoea

This is a term used to indicate that breathlessness is relieved by the upright, as opposed to the lying down, posture It is a common manifestation in severe cardiac failure and it is now generally thought that this effect results from decrease in pulmonary congestion.

Periodic Breathing

This is a frequent phenomenon in the late stages of cardiac failure of elderly subjects and especially when associated with arterial disease high tension, and after morphine has been administered It consists of a regular waxing and waning of breathing, and waning usually proceeds to actual apnoea The phases are each of 15 to 30 seconds in duration, or even longer Where long the patient generally becomes drowsy and difficult to rouse in the apnoeic phase and in the true hyperpnoeic phase he is restless, even to the point of leaving his bed The pulse may vary conspicuously with the periods, but the direction of change is inconstant

The causes of this type of breathing are still obscure, among other factors concerned, deficient blood supply to the brain is suspected

In the next two chapters we shall consider the manifestations of systemic venous congestion, and those of pulmonary congestion and associated troubles separately In a chapter which follows these we shall deal with the main causes of cardiac failure and begin to differentiate different types of cardiac failure as they are displayed clinically.

CHAPTER II

SYSTEMIC VENOUS CONGESTION

THE present chapter describes the symptoms and signs in cases of heart failure in which the systemic venous system is congested

SYMPTOMS

Breathlessness —Because the exceptional case of systemic congestion presents little or no breathlessness at rest, it is difficult to regard this symptom as directly dependent in appreciable measure upon this congestion As has been said it is ascribed chiefly to associated engorgement and other encroachments upon the lungs Be that as it may, patients with systemic congestion of cardiac origin always display breathlessness on exercise, and almost always at rest In cases of deep congestion it is the chief source of distress the breathing is hurried, though usually below 30 per minute it is irregular in rhythm and not in general forcible

Fatigue and exhaustion —Fatigue is an early symptom of the later stages of cardiac failure, but is not used in diagnosis During walking exercise the patients feel their legs heavy, and on reaching home are abnormally weary Many, while lying in bed, feel fatigue constantly Another symptom coming on during exercise is a sense of sinking, and weakness, associated with tremulousness a symptom often termed exhaustion

Fulness of head —A tense feeling across the forehead is a usual symptom of advanced venous congestion, headache, though not the rule, may replace it

Pain —Many of these patients complain of aching over the

precordium, and when hepatic congestion is deep, continuous aching pain of a distressing kind develops in the right hypochondrium

Vomiting is not uncommon after food has been taken and sometimes interferes with the oral administration of drugs

SIGNS

The first clinical signs of general systemic congestion are to be found in the systemic veins, rather than in the legs or abdomen Study of the veins still suffers an unfortunate neglect, in these vessels are to be found some of the most valuable signs we possess in managing heart cases A full grasp and working knowledge of the phenomena to be discussed cannot be attained at once, but only by diligent observation and thought

MEASURES OF VENOUS PRESSURE

Direct method and principle—Direct measurement of venous pressure will often provide an invaluable guide to a patient's state, repeated readings will often indicate clearly the course of the malady But the direct measure can never come into general use, moreover, it can be replaced by simple bedside tests that any thoughtful observer may apply The direct method will be described briefly and mainly with the object of illustrating the hydrostatic principles involved The apparatus in simplest form consists of a U tube manometer, connected to a wide needle by rubber tubing, the whole system being filled with a solution of sodium citrate to prevent clotting The needle is driven into a vein, and the height at which the fluid comes to rest in the manometer is observed A vertical measure, taken from the meniscus to the point at which the vein is punctured accurately measures the pressure in the vein lying at its then level If the vein is in the arm and the arm is lifted up, the pressure in that vein will fall, or if the arm is lowered it will rise, correspondingly, thus, measuring the actual pressure in the vein has little value, since it is constantly varying in different circumstances But if there is no local impediment in the venous system between the chosen vein and right auricle, the veins being widely patent, then the meniscus of fluid in the manometer will remain at the same height relative to the heart, whether the arm is raised or lowered Thus, the height of the meniscus serves as a gauge of

pressure in the right auricle, it is this gauge that is of value If the needle is passed into the median basilic vein of a normal subject lying supine, the fluid column in the manometer will usually come to rest level with the sternum, or a centimetre or two below it, or less commonly above it Thus if the punctured vein lies level with the sternum the meniscus will come to rest at or about the level of the puncture and as the arm is depressed, it will not move appreciably relative to the sternum, but the column will reach above the vein by the amount the arm is lowered The venous reservoir thus behaves much like a simple reservoir (Fig 1) In this the surface of fluid lies at a given level and there the pressure is atmospheric or zero In the depths of the reservoir pressure is greater, and it is

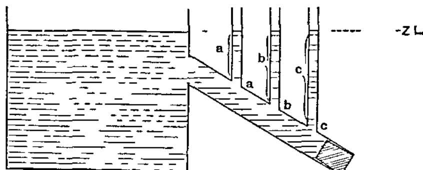


FIG 1—A reservoir of water has a wide pipe joined to it If manometers are attached at *a* *b* *c* the measured pressure above these points is represented by the increasing size of the columns *a* *b* *c* but all these columns rise to the same zero level (Z L) All indicate equally well the height of the water in the reservoir

greater by the amount of the vertical column of fluid that extends from any given point to the surface of the fluid

In determining venous pressure by bedside means, attention should first be directed to the level at which the veins collapse

Venous swelling and collapse—We may use the term zero (or atmospheric) pressure level of the fluid in the venous system in much the same sense as it has previously been employed in speaking of the reservoir, and that level is near the lower border of the manubrium sterni in normal subjects This point of reference is chosen because it represents approximately the level of zero pressure whether the body is horizontal or vertical, or in any intermediate position It is likewise true of the flaccid body that the pressure in veins lying at different levels below the sternum is greater and at levels above the sternum is equal to or less, than atmospheric pressure Thus normally, all veins lying higher than the manubrial point are

collapsed, all lying below it are distended If, therefore, we can gauge the precise level at which the veins collapse, we have a gauge of [illegible]

These, when the arm is warm and hangs limply by the side, are swollen, let the arm be lifted in its flaccid state, while the veins are watched and, in normal resting people, these will flatten as the hand comes to the height of the manubrium The arm must be lifted passively, it must not lift itself, for active movement brings the muscles of the limb into play, and these press upon the veins and disturb the test It is better to use the cervical veins as a routine, for their course to the heart is shorter The subject is stripped and laid upon his back the head resting upon pillows (Fig 2) with the parts concerned as flaccid as they can be made The external jugular veins are then usually to be seen as swollen vessels in the neck, but, as they are traced upwards, the swelling ends, it ends at a point that represents atmospheric pressure in the veins, and in normal people this is at a point of the neck that is level with the sternum, or a little higher or a little lower

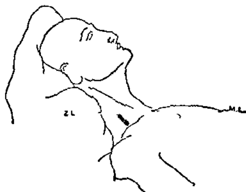


Fig 2 —Normal subject supine with head on pillows The zero level (Z L) indicated by the top point of swelling of the external jugular vein lies a little below the manubrial line (M L)

It is necessary to be sure that the swelling ends where it seems to in the external jugular, and that the vein is not merely running deeper in the higher part of its course To find out press a finger lightly on the vein below and it will at once fill in its length and show its whole superficial course The natural swelling of the veins will extend perhaps a third of the way to the jaw, more or less, according to the inclination of the neck to the body Lift the foot of the couch and in the neck the blood tide advances, lift the head of the couch or the shoulders and head on extra pillows, and it recedes In these acts think of the surface of the venous pool as

you would think of the level of fluid in a long pan of water, lifted at one end or at the other, watch the rise and fall so that you may recognise the surface and know the general level (Fig 3)

The venous zero level rises to various heights in congestion Where venous pressure is raised a little the column is seen to stand in the neck of the supine subject as high as the centre of the sterno mastoid muscle or beyond this, it comes unmistakably above the sternal level on both sides of the neck Where pressure is greater, the veins are swollen to the jaw Note especially that it is not the anatomical point reached in the neck that matters, but the vertical

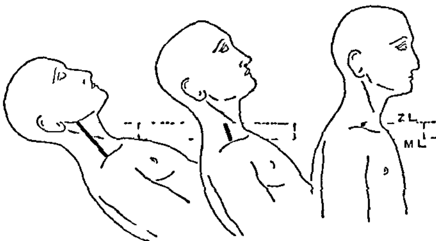


Fig 3 —A diagram illustrating excessive venous pressure equivalent to about 8 cms of water In the upright position the zero level may be just a little above the clavicle as the subject is incl ne- the zero level comes to occupy a higher and higher position in the neck until it reaches to the angle of the jaw Its vertical distance above the manubrium does not change much

distance above the sternum The former is affected by the inclination of the body

Where pressure is higher still, the veins remain swollen through out the neck when the head and shoulders are lifted off the bed The gauge remains unaltered, it is the vertical height to which the swollen veins extend, or may be raised, above the sternum without collapsing The veins are never full in the neck in a normal unclothed person standing easily in ordinary circumstances in the erect posture, in conspicuous congestion they run the length of the neck like cords

Veins swollen only on one side point to local obstruction of their immediate outlet and have no value from our present standpoint, often such swelling can be released by a little rotation of the neck,

the vein that has the lowest pressure is alone to be used as an index of general venous pressure

The bedside method here described of estimating the level of venous collapse is not wholly adequate because in many patients superficial veins of sufficient size to stand out from the neck are not found In such cases, and they are frequent, the required information is often to be obtained by closely observing the venous pulse and its behaviour

Venous pulse as index—Hitherto we have treated the venous reservoir as though the zero level were unvarying Actually this is rarely so, almost always the level fluctuates a little with the beat of the heart, and this fluctuation is often very helpful in gauging the level to which the veins are filled The venous pulse as we see it in the neck is a normal phenomenon, being caused by alternate expansion and collapse of the vein in which it is occurring, and this expansion and collapse may be regarded, properly, as resulting from the rise and fall of the blood tide in the veins Though it occurs at lower levels also, the venous pulse is of greatest amplitude immediately at the general level of venous collapse Veins that are tensely distended cannot pulsate appreciably, neither can those that are empty It follows that pulsating veins are full veins, and that the uppermost point of free pulsation is an index of the level to which they are filled, this index is used, as previously remarked, when the actual point to which the veins are filled cannot be observed directly Thus it is clearly most important to be able to recognise venous pulsation when it is seen but this requires careful study if it is to be accomplished with certainty

In normal health, the subject being supine, venous pulsation occurs at the root of the neck often, and especially in young subjects, it is a movement of large amplitude It is obviously a venous phenomenon when seen in such a superficial vein as the external jugular In superficial veins it can be studied in relation to the level of venous collapse, in the stretch below the point of highest filling the vein pulsates freely, the movement being complex and undulatory (as in Fig 4) In these the level of pulsation is seen to alter with respiration, approaching nearer to the heart at each inspiration, for this lowers venous pressure But there is also a diffusely distributed venous pulsation in the lower neck occurring along the line of the carotid sheath and subclavian vessels This is mainly produced in the internal jugular, in the subclavian vein, and in the large superficial tributaries of the latter in the subclavian

triangle Most of the pulsation in the neck is of such deeper origin and, while it cannot then be seen to arise from veins, can and must be recognised as venous by its characters, its usual softness, its frequent large volume, its general fall with systole and rise with diastole, the undulations upon it, and its change of site with posture Venous pulsation disappears altogether when the erect posture is assumed, because the cervical veins then collapse, it moves higher up the neck when the head is lowered or the abdomen is pressed upon It extends relatively high in the necks of young people Such in general is venous pulsation in the uncongested

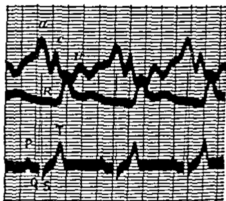


FIG 4—Venous (or jugular) radial and electrocardiographic curves from a normal subject The [illegible] and marks the beginning of ventricular systole in the venous curve The *a* wave lies in presystole and results from the auricular contraction The summit *v* marks the end of ventricular systole and occurs [illegible] events are later in the mechanical records than in the electrocardiograph by a time interval of $\frac{1}{10}$ sec or more This delay is due to time lost in transmission of the waves from heart to neck or heart to wrist and in the rubber tubing

In the congested, the signs are modified The field of pulsation is displaced to a higher level in the neck The largest pulsation comes from the deep lying internal jugular and, with the patient's head or shoulders supported on pillows, frequently extends to the jaw and moves the lobe of the ear It is often slow, welling, and sustained, rather than undulatory When much congestion is present, and the subject's head is well raised on pillows, the veins are often too tense to pulsate, tilt the patient into a more erect posture, and the level of zero venous pressure falls in the neck and, as this tide recedes, abundant pulsation appears below the jaw In these patients venous pulsation may be seen in the neck when they stand, or spreading to the veins of the upper arm or forearm while they lie Whenever veins freely pulsate appreciably above the level of the manubrial line they are overfull, the higher the level of pulsation the greater the overfilling It is not venous pulsation that is abnormal, it is

the high level at which it occurs that constitutes abnormality The examination must be made thoughtfully, thus in many normal subjects, if they lean forward as they sit, prominent pulsation appears in the jugular veins that is so because pressure on the abdomen in this posture displaces blood into the neck

Venous is very often mistaken for arterial pulsation especially by those who think the distinction always easy, it is a serious blunder to make It is not generally recognised that venous pulsation is much the commoner, has usually the greater amplitude and more often moves the lobe of the ear Pulsation that is maximal in the subclavian triangle is almost always venous pulsation maximal in the carotid triangles often is An arterial pulsation is usually maximal in the carotid triangle and it increases when the erect posture is assumed a corresponding venous pulse lessens or disappears under the same test An arterial pulsation that is visible is always palpable it jerks sharply against the finger to a sustained thrust Venous pulsation is very rarely strongly palpable and only occasionally can be felt the testing finger should be laid as lightly as possible upon it When as during congestion, the venous pulsation tends to lose its undulatory character and becomes more sustained and sometimes plainly palpable the direct distinction between it and arterial pulsation is not always easy, but in such cases the remaining signs of a congested venous system are always manifest

OTHER SIGNS OF SYSTEMIC CONGESTION

The liver —The earliest sign of a congested liver is usually obtained by percussion I prefer to use it in the mid line and of moderate intensity norma
midriff the thin margin of
fails materially to modify
light percussion is used In congestion enlargement is
the firm smooth margin is usually palpable at a
between rib margin and navel or lower failure to pal
due to beginning to feel at too high a level When
enlargement the abdomen is usually convex between
umbilicus and the liver pulsates softly like the vein
often present though not always easy to detect It ma
sometimes when the patient lies on the left side Th
abdominal wall over the enlarged liver is frequent
associated with rigidity and pain In very deep and pr

gestion the skin becomes jaundiced except where it is involved in oedema, in such regions the skin shows no stain, owing to dilution of the bile pigment, which passes the capillary wall less easily than the fluid Jaundice, whether cardiac or frankly obstructive, can be cleared from the skin by impeding venous flow for a few hours with a rubber bandage, it returns quickly when the bandage is removed Jaundice in congestion is sometimes related to pulmonary infarction

The kidneys —The normal quantity of urine passed daily is 30 to 50 oz (1000 to 1500 c c) it varies much with the activity of the skin and the amount of fluid drunk Congestion of the kidney gives a scanty and concentrated urine which deposits phosphates and urates and may contain a trace of albumen, a few hyaline casts, and red blood corpuscles

Cyanosis and oedema though frequent accompaniments of venous congestion, are met with in many other conditions While these may be guiding signs leading to the diagnosis, neither can be safely used as an evidence of venous engorgement It will be convenient to consider each of these phenomena at length a little later (Chapters IV and VI)

Malnutrition is a common accompaniment of long standing venous congestion Often when the weight has risen owing to accumulation of fluid in the tissue spaces, the face, arms, and chest show obvious wasting

DIAGNOSIS CORRELATIONS AND DISCREPANCIES

The liver —When the full signs of systemic congestion are present, neither close observation nor much discrimination is required to know it The diagnosis of early congestion is more difficult and in general much more important The diagnosis of congestion whether early or late should finally be made almost exclusively on the evidence provided by the veins and the liver Experience teaches that to place reliance upon a single sign is precarious Compare this sign and that and confident recognition of the patient's state grows as these signs group themselves together to form a harmonious picture Thus on finding unquestionable evidence of engorged cervical veins we look at once at the liver and finding it enlarged, are satisfied that the engorgement is general and not confined to superior caval territory

Generally speaking enlargement of the liver in its several degrees goes hand in hand with engorgement of the veins The liver does not

usually enlarge before the rise of pressure in the veins can be detected, neither do the veins swell without the liver edge descending perceptibly Thus, the early diagnosis of congestion turns upon a close examination and correlation of these signs

The differential diagnosis of congested liver from other forms of enlargement rests mainly on an examination of the veins The differential diagnosis of acute congestion is dealt with later (page 86) If it is beyond question that the veins of the neck are bilaterally congested and it is equally beyond doubt that the liver is not enlarged, an obstruction of the superior cava must be considered Its diagnosis will rest upon the discovery of anastomotic veins and upon failure to induce the veins of the neck to pulsate A second reason for the same discrepancy is atrophic cirrhosis of the liver in a congested patient, the diagnosis of the liver condition will then turn upon the degree of hardness, and perhaps irregularity, of the liver margin A third reason for the discrepancy is vascularity of the thyroid gland (page 264)

There is the reverse case an engorgement of the liver has been present for a long time and the venous spaces within have become permanently dilated and its substance fatty or fibrosed In such, even if the signs of increased pressure in the veins greatly decline, the size of the liver may not decrease much or proportionately It is a discrepancy which previous knowledge of the course of the malady explains

Breathlessness —Here is another important clinical correlation Patients who suffer from cardiac failure with systemic congestion are without exception breathless, either while lying flat at rest or upon very slight exertion The cause of such breathlessness is at present beside the point We are dealing with a useful clinical correlation When early congestion is strongly suspected, but not quite certainly diagnosed, then the presence of such breathlessness goes far to complete the diagnosis If, on the other hand, a subject is thought on examination to manifest early signs of venous stasis, and yet it is clear that breathlessness is not experienced even with quiet or moderate exercise, the opinion first formed must be revised, for it is incorrect Moreover, in failure there is in general a relation between the degree of breathlessness and the pressure in the veins, if it is established that a patient has no congestion of the venous system and is yet definitely breathless in bed, then that breathlessness is rarely cardiac in origin There are instances where systemic congestion predominates over breathlessness, and other and more

numerous instances where breathlessness predominates over systemic congestion Recognition of this or that predominance is of high diagnostic value, they are more fully discussed in Chapter V The proper use of such knowledge saves a number of serious diagnostic blunders It is to be repeated that a full understanding of cardiac failure is obtainable only by studying the manner in which its symptoms and its signs are linked together, the subjective and objective manifestations are part and parcel of one process, and dependent upon the same fundamental cause, namely, lowered capacity of the heart for work

CHAPTER III

PULMONARY CONGESTION, OEDEMA, AND INFARCTION

Chronic Passive Congestion and Oedema of Lungs

Passive congestion of the lung is due to increased pressure in its vessels This can be caused by anything that obstructs the outflow of blood from the lungs Thus a chronic state of pulmonary congestion results when the pulmonary veins are compressed or thrombosed, or in such narrowing of the mitral orifice as raises the average pressure in the left auricle very appreciably It is the rule to find pulmonary congestion when the heart as a whole is failing, this association with systemic venous congestion has been noted already It is particularly to be associated with preponderant failure of the muscles of the left ventricle beating against increased resistance, for with the right ventricle working efficiently and filling the lung vessels the left ventricle is then unable to eject blood fast enough to keep the pressure in left auricle and pulmonary veins down to the normal level

In passive congestion transudate from the small engorged vessels is increased, making the tissues a little, and in more profound forms manifestly, oedematous

Engorgement, and an increase of fluid within alveoli and bronchioles, puts many alveoli out of effective action, the blood passing through to the left heart and systemic arteries without changing its venous state

When of very long duration congestion widens the pulmonary arteries, and these often become atheromatous with the engorgement of vessels the bronchi are a little inflamed the connective tissue of the lung generally is diffusely increased, and the alveoli contain numerous phagocytes, a condition termed "brown induration" of the lung

SYMPTOMS AND SIGNS

The chief symptom of pulmonary congestion is breathlessness at rest The dyspnoea is often extreme the breathing is rapid, the rate

often surpassing 30 or 35 per minute Orthopnoea and irregular breathing is the rule

Oedema brings cough with at first a little sticky ultimately watery pink or obviously blood stained sputum which may become copious and contain a high protein (2 to 3 per cent) content

Cyanosis is the rule and often deep it is of central type (page 47) High pressure in the pulmonary artery is often accompanied by a ringing 2nd sound at the pulmonary cartilage such accentuation however may not be used as certain evidence of such increased pressure (see page 233) Dilatation of the main pulmonary artery may be *identified by X ray (Fig 30 page 151) Dilatation of its* branches increases the width and density of the root shadows to which dilatation of the veins contributes when the right pulmonary artery is dilated it throws a short thick horizontal shadow from the end of which that of the chief branch runs downwards to give a characteristic invert L shaped shadow often exhibiting unusual expansile pulsation The lung fields especially their lower parts are abnormally dense These appearances are often complicated by the shadow of basal pleural fluid and by lung oedema

In congestion the breath sounds at the bases of the lungs are often impaired and the percussion note dulled Oedema adds to the prominence of these signs and brings at first and inconstantly subcrepitant rales but if there is more exudate the rales are numerous bubbling and often noisy Often the picture is complicated by the simultaneous presence of infarction or of terminal bronchopneumonia

The arm to tongue circulation time in congestion is in the rule increased This is taken by timing the interval elapsing from an injection of sodium dehydrocholate (5 c c of 20 per cent sol) or saccharin (2 5 g in 2 c c water) into the antecubital vein to the signalling by the patient of a bitter or sweet taste in the mouth The normal interval at rest is 8 to 16 sec but in pulmonary congestion it may be prolonged to 30 45 or more sec The arm to lung time measured by injecting 0 2 c c of ether in 1 c c saline into the vein and noticing the first appearance of ether in the breath is normally 4 to 8 sec this interval may be unaffected Thus the delay in the arm to lung time seems to be in the pulmonary veins

Acute Congestion and Oedema of Lungs

ACUTE CONGESTION WITH OEDEMA (CARDIAC ASTHMA)

The term cardiac asthma is used to indicate a special form of severe paroxysmal breathlessness occurring mainly at night The chief clinical associations are detailed on page 32

The attack —An elderly subject, having few symptoms or experiencing a little breathlessness while resting or on effort in the daytime, retires to bed and, lying recumbent, sleeps He wakes in the early hours feeling oppressed and breathless, sits up in bed and pants, becomes restless as breathlessness increases, is distressed and sits on the edge of the bed or moves to the window; after a few minutes or longer he becomes wheezy and soon the attack subsides

The severer attack wakes the patient and brings him at once to a sitting position with a sense of intense suffocation The breathing is increased in rate and becomes more and more forcible, he clutches surrounding objects and brings all the accessory muscles into play But the chest increasing in size moves less and less effectively Distress is terrible Little air can be taken into the lungs and expiration is prolonged and powerful Cyanosis comes and quickly deepens while at first the veins may remain unengorged Pallor may be added Sweat breaks out and is profuse The man is brought within a very short time into the throes of a fierce struggle for breath The chest is resonant and soon filled with adventitious sounds The patient becomes semi conscious The attack begins to subside In this, and sometimes in the milder, attack cough develops and the patient brings up a little frothy and usually blood stained sputum He is left exhausted Such a severe attack may last a half hour an hour, or more Recovery is almost invariable but it threatens life, and occasionally ends in fatal oedema of the lungs In the early stages the pulse is quick (up to 120) and strong and the systolic blood pressure is found to have risen In the later stages pressure may fall and the pulse become almost imperceptible The X ray signs are those already described of congestion with or without oedema

Short attacks may be repeated one or more times in a night Similar attacks also occur occasionally when patients lie down by day sometimes they are provoked by exercise

The attack is directly due to a rise of pressure in the pulmonary vascular system producing turgescence and oedema of the lungs

It is believed usually to result from temporary failure of the left ventricle It has been suggested that this is provoked by an increase in blood volume more tissue fluid being drawn into circulation when the subject lies down Such an increase is known to occur, and with this we may couple the decline in the volume of the legs which happens in both normal and abnormal subjects during the resting period of night

Diagnosis —The attacks of breathlessness here described are mainly nocturnal They must be distinguished from attacks brought about differently Patients suffering from failure with congestion and orthopnoea slip down in bed off the pillows in their restless sleep after a time they wake breathless and distressed This form of breathlessness can easily be remedied if recognised Patients suffering from periodic breathing are sometimes wakened repeatedly at night by particularly long and violent hyperpnoeic periods

ACUTE PULMONARY OEDEMA

In acute oedema the lungs are not only congested but, at their bases or throughout are heavy and have lost their spongy crepitant quality They *are firm, pale and pit on pressure* When the lung is cut a thin blood stained fluid runs from the surface and, when it is squeezed, this fluid runs freely and is frothy The same fluid is found in the respiratory passages It is believed usually to be a con sequence of primary failure of the left ventricle A similar condition of the lungs can be produced experimentally, and acutely, by inter fering mechanically with the action of the left ventricle, the right continuing to beat efficiently

Acute and severe pulmonary oedema can occur in single or re-current attacks The chief clinical associations are given on page 32

The onset may resemble exactly that of the attacks previously described, occurring usually at night and waking the patient from sleep or it may come in the hours of daylight and wakefulness At the beginning of the attack systemic blood pressure has been found raised The separation of these attacks from those previously described is in fact arbitrary and necessitated only by relative dry ness of the latter and their frequent repetition In frank pulmonary oedema, while breathlessness is usually urgent from the start cough is more prominent and frothy blood stained fluid is freely or profusely expectorated The march of events is often speedy Cyanosis deepens rapidly, breathing becomes progressively more

urgent and less effective The attack may last at its height for hours Recovery is the rule, but severe attacks are very dangerous In fatal attacks the pulse becomes steadily weaker until imperceptible, the veins swell, and the breathing becomes more gasping less frequent, and then weaker until it ends

In a fulminating form, the oedema occurs so rapidly and is so intense that, within a minute or two of crying out in fear, the patient is drowned by the copious blood stained fluid that pours into the respiratory passages and overflows frothing from the mouth and nose

Pulmonary Infarction

Infarction of the lung usually originates from an embolus and the commonest source is a clot dislodged from the main veins of the lower limbs and from those of the pelvis When the clot arises in the heart itself, the original thrombus is attached to the wall of the right auricular appendage or less commonly at the apex of the right ventricle or from the veins The detached clot is swept ultimately into the pulmonary artery, entering and blocking one of the pulmonary arterial branches Frequently thrombus accumulates upon it subsequently *Infarction is occasionally due to thrombosis* originating in a pulmonary artery or pulmonary veins The blocking of a pulmonary branch leads to haemorrhagic infarction of the corresponding lung territory Several areas may be infarcted simultaneously by separate clots Base or apex of the lung may be involved The infarcted areas are usually wedge shaped, the base involving the visceral pleura the pleura becomes covered with [illegible]

[illegible]

clots in the auricles is encouraged by the paralysis of the auricular wall which fibrillation induces their detachment is favoured by active movement of the wall when the auricle is beating normally *Consequently embolism* is especially apt to occur when the auricles resume their beating after they have been fibrillating, as in paroxysmal fibrillation, or at the resumption of normal rhythm under quinidine Infarction occurs also in cases of mitral stenosis in which the heart's mechanism is unchanging, these cases are as often found

in man is temperature, the higher the temperature of the part the faster the exudate occurs, a clinical instance is that in which dropsy of the legs forms quickly in predisposed patients while they sit before a fire.

Recognition

In cardiac dropsy much of the fluid collects in the subcutaneous tissues, where it is easy to identify It causes swelling, and the overlying skin is stretched and becomes as a consequence pale and shiny If the stretching is very great "lineae atrophicae" may develop, in other cases the skin oozes, or it reddens, and blisters appear Oedema is frequently accompanied by tenderness, which may outlast the remaining signs when oedema disappears In recognising early dropsy of the subcutaneous tissues it is usual to press upon the part with the finger, the excessive tissue fluids are thus displaced and the skin indented as is plastic clay when similarly treated Firm pressure continued for 5 seconds will often disclose a slight oedema that would otherwise escape notice Even so elicited, pitting is not a very early sign of the increase in tissue fluids It is known that the volume of a limb must increase, through the accumulation of tissue fluids, by a tenth, before pitting becomes clearly recognisable Patients who are becoming dropsical begin to put on weight some days before pitting can be found, patients who are losing dropsy continue to lose weight for some days after the last sign of pitting has gone Advanced dropsy is at once recognised by the relative hardness of the tissues and inability to pick up the skin The extent of such dropsy can be traced quickly by simple palpation.

Distribution

Cardiac oedema is one form of dropsy that is essentially hydrostatic in its distribution In patients who spend much of their time upon their feet or sitting, the feet are the first parts of the body to swell and they become most swollen It is in the instep and ankle that oedema is usually first seen in such patients, though the distribution may be modified by the nature of the footgear Patients with early dropsy who have been up and about often find the oedema forming as a rim over the upper parts of the shoe or boot, swelling of the foot follows removal of outdoor shoes, which are then replaced with difficulty In patients who lie in bed pitting is often found first just behind the malleoli A continuous and gradual involvement of the limb from foot to thigh, genitalia, and trunk

occurs in those who spend their time sitting In bed patients, after the ankles swell, the genitalia or sacral region may next show change These differences depend upon the levels at which the several affected parts lie If the buttocks sink low in the bed, sacral oedema will come earlier and be the more in evidence Parts upon which the patient actually rests show little oedema, the fluid being readily displaced by pressure, when patients are propped on several pillows, and oedema has risen to the trunk, it is common to see a ridge of oedema across the back, between the areas pressed upon by two separate pillows Oedema spreading on the trunk above the navel is relatively uncommon in purely cardiac cases, occurring only in very advanced stages of failure Similarly, it is unusual in the hands, unless these are kept for long periods hanging down, or unless, as sometimes happens, thrombosis occurs in the main veins of one limb, oedema being then unilateral Oedema of the face or scalp is rarely cardiac, but usually renal

To retain a clear idea of the influences that affect the distribution of cardiac oedema is important from several points of view Oedema cannot be decreased by changing the patient's posture, but it can be redistributed If a patient comes with slight dropsy of the feet and is placed flat in bed, this oedema will often disappear within a day or two because the excessive fluid in the tissue spaces of the legs moves to tissue spaces higher up in the body Hanging the legs down often enables these limbs to act temporarily as a reservoir to accumulate fluid as a preliminary to drainage (page 41)

Differential Diagnosis

Dropsy of cardiac origin is recognised by its hydrostatic distribution and by its association with increased general venous pressure If by careful use of the methods described in the preceding chapter general venous pressure is judged to be normal, then dropsy in the legs can be stated to be due to no weakness of the heart This very valuable rule is broken only by those instances of cardiac dropsy in which owing to an unusually rapid fall of venous pressure, as from venesection the disappearance of oedema lags notably behind Dropsy of hydrostatic type is a very common affair, and much more often than not it is produced in other ways than through cardiac failure It may result rarely, from various forms of local obstruction of the inferior vena cava or its tributaries, as by thrombosis, by pressure from tumour or collections of fluid in the abdomen It is

As knowledge of heart failure has developed stress has come to be placed more on the state of the cardiac muscle, for it is quite clear that the muscle may be so damaged that the heart will fail, even at a time when the burden it has to bear is no more or is even less than customary A simple instance is that of failure with congestion arising while the subject is at rest, from thrombosis of a coronary artery killing a substantial portion of the ventricular wall Another instance is pernicious anaemia, through which nutrition of the heart muscle as a whole may be so reduced that failure supervenes The acute infectious diseases provide examples of acute heart failure resulting purely from poisoning of the muscle

In asphyxia from any cause, the heart swells and the veins become more and more engorged until no blood is ejected, an acute failure due directly or indirectly to lack of oxygen, but one in which increased resistance to output may for a time play a minor part In subacute bacterial endocarditis (page 190) and in hyperthyroidism (page 264) heart failure is chiefly due to change in the muscle, though contributed to by valvular disease or by increased output Another undoubted and frequent example of the same kind is failure with congestion arising during the course of rheumatic carditis (page 201) the fact that such congestion comes, and later disappears completely shows that it is not due in the cases that display this sequence of events to permanent anatomical changes, such as mitral stenosis or pericardial adhesions, for these remain after the congestion has cleared away

It will be found by actual enquiry or observation that patients who develop failure with congestion, while under supervision for heart disease do so more often in immediate sequence to an infection than in response to a period of unusual or unwise bodily exertion the infection is not necessarily rheumatic or pneumococcic, but may consist of a febrile cold, influenza, or bronchitis

One of the most frequent determining factors of congestion is the onset of very rapid heart action, as in paroxysmal tachycardia or in auricular fibrillation (pages 83 and 93) It is most important that the manner in which rapid action affects the heart deleteriously should be understood thoroughly It does so in two ways First it causes the ventricles to expend more energy At each of its beats the ventricle must raise the pressure within its cavity to arterial pressure before it begins to do effective work Other things being equal the waste of energy that occurs in this way increases proportionately to the rate of beating Secondly, rapid beating shortens

the time between beats for recovery, and this not only absolutely but relatively Thus, if the ventricle is beating at 60 per minute, systole has a length of about $\frac{1}{3}$ and diastole $\frac{2}{3}$ second, while, if the rate of beating is 120 per minute, systole and diastole each occupy about $\frac{1}{4}$ second Thus the relative length of diastole is reduced to one half its former value, the ventricle enjoys but three quarters its previous total resting periods But the reaction of different hearts to the strain of paroxysmal tachycardia is very variable The normal heart bears the burden well, it is the weak heart that gives way and allows congestion to develop

It is essential to the treatment of certain types of cardiac failure, and especially to prognosis in cardiac failure that ideas as to causation should assume a proper balance, that the condition of the ventricle should be taken into account as well as the work it has to do, and that, because an increased burden is known to exist, this should not come automatically to be regarded as the whole, or necessarily the chief, trouble That weakened muscle is often the sole or chief, and probably almost always a considerable, factor in causing congestive failure, is not merely a view of academic interest but one that greatly influences the general attitude to heart cases and their management, especially it emphasises the need of guarding these patients against infections of any kind

Cases of chronic heart failure fall into three main causative categories, namely rheumatism (about 25 per cent), hypertension, and coronary arterial disease (alone or together about 50 per cent), the remaining cases are chiefly syphilitic, thyrotoxic, grossly anaemic, or have pulmonary disease

Types of Failure

A real difficulty that arises in any attempt to discriminate types of cardiac failure is that no simple subdivision presents a true or adequate picture Contemporary writings, though rightly distinguishing tend to isolate left and right heart failure It is true enough that first the left or the right heart may fail and the deficiency of one or other may continue to dominate, and the lack of balance becomes established But the patients cannot be divided sharply into two groups the right and the left, for in the great majority both right and left weakness are present simultaneously, though in different degrees It is necessary to recognise at the outset that cases legitimately regarded as instances of pure right or left failure are uncommon, but it is also necessary from the practical standpoint to

differentiate between common types in which right or left failure predominates It is impossible sharply to establish types each having its own distinct causative and symptomatological characteristics this can be done usefully only upon broad lines It should also be understood that any such descriptions must be more or less arbitrary because though pressure in systemic veins can be measured that in pulmonary veins cannot, and so accurate comparison between the degrees of the two engorgements is rarely possible

BILATERAL FAILURE

Many cases of chronic heart failure belong to this clinical class It is seen most clearly in the rheumatic hearts of the early decades but is by no means confined to such The cases present simultaneously the manifestations of systemic venous congestion described in Chapter II and those of pulmonary congestion described in Chapter III Thus the systemic veins and liver are engorged and the concomitant manifestations of renal congestion and excessive tissue fluid are found But they also display breathlessness at rest, and X ray and other signs indicative of chronic pulmonary engorgement.

PREDOMINANCE OF LEFT FAILURE

These patients are usually between 50 and 70 years of age In this age period it is the commonest type of heart failure Men are much more often affected than women Three fourths of them have or have had hypertension with or without coronary arterial disease Many of the remainder have advanced arteriosclerosis with coronary involvement In both groups the coronary involvement may have

majority there is no evidence of disease of the valves but enlargement of the heart is invariable

In these patients breathlessness and orthopnoea are unusually conspicuous and often extreme and the signs of pulmonary engorgement are invariable and often fully displayed periodic breathing is common and so is cardiac asthma Venous engorgement may be moderate slight or even absent The skin of these patients is often relatively bloodless so that cyanosis is concealed Gallop rhythm is usual, alternation of the pulse or gross anomaly of the electro

cardiogram often bear direct witness to faulty cardiac muscle The type is further referred to on page 236

PREDOMINANCE OF RIGHT FAILURE

These cases are rarer than either of the preceding They are notable for the intensity of systemic venous engorgement while breathlessness is in little evidence at rest Such patients may even be able to lie flat without discomfort A deep colour of the skin, especially the facial skin, is usual, and for this reason they over display cyanosis The X ray shows little change in the lungs The electrocardiogram presents signs of right axis deviation (page 124)

The most demonstrative cases of this type are instances of constrictive pericarditis (see page 182) and of cor pulmonale (see page 259) but occasional cases of mitral stenosis display this picture, as do cases of pulmonary stenosis

Very rare cases regarded as pure right sided failure have been described, in which this picture is associated with X ray evidence of enlarged pulmonary artery and conus, accentuated 2nd pulmonary sound and conspicuous signs of right sided hypertrophy, in the absence of valve or pulmonary disease, or of pericardial thickening The pathogeny is obscure

DIFFERENTIATION

The diagnosis of these different types is not difficult in most instances, though, owing to the occurrence of every transitional form, anything like precise differentiation is often impossible The quickest clinical method is to note as a routine the relation between the degree of breathlessness and of systemic engorgement A departure from the usual relation in the direction of excessive breathlessness on the one hand, or that of excessive systemic congestion on the other, should first suggest as a possibility predominance of left or right failure, respectively

Differentiation of the breathlessness of effort syndrome and of nervous breathlessness is dealt with on page 173

ACUTE PULMONARY OEDEMA

The clinical associations are not unlike those in predominance of left failure, the cases being usually elderly hypertensives, subjects of coronary disease, or occasional cases of syphilitic aortitis, or

mitral stenosis, the last particularly in pregnancy It may happen as an immediate sequel to coronary thrombosis and occasionally to an attack of angina pectoris An instructive association is its occurrence in the attack of those rare cases of paroxysmal hyper tension, which are thought to result from release of natural adrenal ine (see page 72) Symptoms, usually slight, of pulmonary oedema may also appear in attacks of paroxysmal tachycardia that bring failure with congestion Acute oedema usually supervenes upon chronic passive congestion, but also upon infarction of the lung

Prognosis

Failure with congestion is among the very gravest manifestations of cardiac disease but it has been said with some truth, though not with accuracy, that no patient dies in a first attack The gravity of the immediate outlook naturally varies with the degree of conges tion, and the presence of general anasarca, fluid in the body cavities or congestion and oedema of the bases of the lungs, constitutes at all times a condition of great seriousness But a prognosis cannot often be made upon the simple basis of signs The manner in which failure has developed requires close consideration Sometimes a state of congestion has followed relatively suddenly upon an attack of acute bronchitis or infectious cold, and in such it will usually subside, leaving the patient much as he was Following upon pneumonia or occurring during chronic rheumatic infection the outlook is less propitious Developing during the course of subacute infective endocarditis it is a terminal event, for here the infection that is its cause is incurable Failure may be caused by anaemia or by hyperthyroidism which when ultimately cured may leave no cardiac weakness it may follow an attack of coronary thrombosis and complete recovery of the former reserves cannot then be expected, though much lost ground may be regained In all these instances we are weighing the chances that a primary weakness of the muscle may disappear with its cause

Congestion may develop as a consequence of rapid ventricular action, at the time when fibrillation or flutter comes, or when the action of the ventricle in response to a fibrillating auricle becomes rapid through neglect of proper treatment Cases of these kinds are frequent and favourable The depth of congestion cannot be used to gauge ventricular strength unless the precise conditions of beat ing are known Of two cases presenting the same signs of failure,

the heart is sounder in the case in which it beats under less favourable conditions, it is the probability of obtaining relief from disadvantageous conditions of work or from an increased burden of work, that makes the prognosis more favourable Thus, given a certain grade of failure, the presence of fibrillation with rapid ventricular action improves the outlook Similarly, a patient who develops congestion while engaged in heavy manual work has a better prospect than one who begins to suffer similarly in a sedentary occupation The development of congestion, having no transient or removable cause, by a subject already in bed, is ominous Patients with preponderating pulmonary congestion or with milder systemic congestion and presenting the phenomenon of cardiac asthma, belong to an unfavourable prognostic class Many of them are elderly, many present other ominous manifestations (see page 237), many are dead within six months, and most within two years, of the first asthmatic seizure

When the signs of failure are severe, the end is often brought by infarction, terminal bronchopneumonia or by the supervention of coronary thrombosis These are ominous additions, as is the appearance of stupor or delirium

But the outlook in most cases of systemic and even of preponderating, pulmonary congestion is by no means that of quick disaster and a final prognosis is rarely justified until reaction to treatment has been tested While continued signs under prolonged treatment are unfavourable yet it is remarkable how rest and above all skilful nursing persisted in for months will bring such patients to relative comfort and safety

It will be evident from what has been said that the immediate prognosis in congestive failure requires deliberation and caution

The place taken by cardiac failure, whether early or late, in a general prognosis that attempts to estimate the duration of life, will be found in other parts of this book, and especially in its last chapter

Treatment

The treatment of failure with congestion is described in the first place without reference to the underlying cause of muscular weakness or to the burden the muscle has to bear Most cases of failure with congestion are chronic, and the principles and measures of treatment are in general the same for all It is convenient to deal first and in detail with cases of relatively severe congestion present

ing normal rhythm, and then briefly with less severe cases, usually presenting auricular fibrillation This will form a sufficient basis, and such modifications and additions as are necessary in special cases will be added in other parts of this book

Indications for the management of patients suffering from the grades of failure that precede and succeed congestion will be found under "After care" (page 43), and later and especially in the last chapter of this book.

SEVERE CONGESTION

Rest —Failure with congestion is one of the few manifestations of heart disease that calls for treatment in bed, and it calls for this imperatively The watchword throughout the treatment of congestion is indeed rest rest for the body, rest for the mind, rest for the heart Heart failure that has reached the stage of showing deep congestion requires skilful handling of the patient by day and night nurses The patient must be guarded against effort of all kinds He should lie still in bed, should not lift himself or change his own position, should not wash, dress, or even feed himself He may be moved a little when uneasy He must be lifted on and off the bed pan, the nurse, therefore, must be strong and will require help The patient should be discouraged from talking beyond indicating his immediate wants, and from attempting to assist the nurse who moves him These precautions against effort may be relaxed appropriately in cases of more favourable type.

The motions should be kept loose by means of salines, so that straining is avoided, two actions daily of the bowels, or one if full, is adequate, purging such patients, even if they are oedematous, is not good treatment

Posture —Congested patients are uncomfortable unless the head is well raised When the head is lowered the subject may be seen to become increasingly cyanotic in the face and breathlessness appears, or in the orthopnoeic case increases to the point of severe distress Attacks of breathlessness at night are often due to slipping down the bed and off the pillows, this should not be permitted to occur

The author's bedstead (Fig 5) facilitates nursing in cardiac failure The wire mattress of this bedstead is in three pieces, hinged together and controlled so that they can be inclined at any desired angles The patient can be propped up to sitting positions by raising the back rest (A) Lifting the thigh rest (B) prevents his slipping,

and this device is more satisfactory than the simpler alternative of tying a bolster across the bed beneath the patient's thighs The two adjustments described, combined with a lowered leg rest (C), con vert the bed into a chair

Sleep —Congested, and especially very breathless patients who fail to obtain a number of hours' sleep in each twenty four hours lose ground through fatigue and weakness The hours of sleep should be reported as a daily routine and, in general, hypnotics are used when the hours of sleep fall below six Hypnotics of the barbitone series are of value, there is a choice between many As

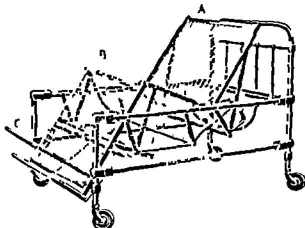


FIG 5.—The author's cardiac bedstead. (See *British Medical Journal* 1928 ii p 977)

in other cases they should be prescribed under official, and not proprietary, names Barbitone soluble is given in doses of 5 to 10 grains (0 3 to 0 6 g), allobarbitone or pentobarbitone sodium in doses of 1½ to 3 grains (0 1 to 0 2 g) A single powdered tablet is placed on the tongue and swallowed with a little fluid an hour before sleep is desired Chloral 20 grains (1 3 g) is a well tried and excellent hypnotic, it occasionally induces nausea or vomiting and should not be repeated in this circumstance Paraldehyde, 2 to 4 drachms (3 5 to 7 c c) with ½ oz of brandy or in capsules is effective, but its taste and smell are objectionable Where sleeplessness is associated with pain or much breathlessness ⅙ to ¼ grain (0 01 to 0 015 g) morphia should be used where it is associated with irritating but ineffective coughing $\frac{1}{10}$ to ⅕ grain (0 006 to 0 013 g) heroin hydrochloride or ¼ grain (0 015 g) codeine phosphate may be given by mouth

Diet —Patients with grave congestion have little desire for food

and often become distressed while masticating Distension of the stomach by food is clearly undesirable The food therefore should be small in quantity, of high nutritive value, and easily taken (see Diet I, page 292) The fluid and salt intake should be restricted in the presence of dropsy A diet of about 800 calories can be main tained for a few days or for a week, without appreciable loss of strength, it may be increased gradually as congestion subsides, or as other circumstances dictate

Venesection —Venesection is a most potent remedy and should be used in most cases in which there is material systemic venous *engorgement* (*8 or more cm excess pressure*) *Only obvious anaemia* contraindicates its use

To ensure relaxed veins and ample flow, the arm, including the hand, should be thoroughly warm The pneumatic cuff of a sphyg momanometer is placed as high as possible on the upper arm, and distended at a pressure of 60 mm Hg a rubber band wrapped around the arm at a tension adequate to distend the veins strongly, but insufficient to decrease the strength of the pulse, is of equal service The obstruction to the veins should be maintained during the period of bleeding This may be accomplished by dissecting out a vein and opening it under local anaesthesia, but is better done by thrusting a needle of large bore through the skin into the vein so as to meet the current of blood with its point towards the hand, in the latter case flow can be expedited from time to time by applying suction with a syringe In an adult of medium weight (140 lb) 10 to 20 oz (250 550 c c) of blood can be taken, according to the severity of congestion Rapid bleeding lowers venous pressure promptly and profoundly, by the simple act of withdrawing fluid from the venous reservoirs A decrease in the fulness of the cervical veins is almost always, and in the size of the liver is often, to be ascertained Patients will almost always speak of relief both from breathlessness and from the sense of tension in the head sleep becomes easier to obtain

In cases that react most favourably, venous pressure remains low after bleeding, oftener it is found to be raised again, though not to its original level, on the succeeding days If the rise in level con tinues, another bleeding of 10 oz , a week or ten days after the first, may be required and in the most obstinate cases three or four venesections may be needed within four to six weeks

In this stage of failure the patient should be visited at least once a day At these visits and during subsequent progress the most

important enquiries will concern breathlessness and the hours of sleep The signs to watch are not in the heart, but in the veins and liver, in the legs, and at the bases of the lungs

Oxygen —It is useless to give oxygen through a funnel held near the patient's nose, the oxygen content of the inspired air is insufficiently raised thereby Delivered through a nasal catheter, passed well back, a much higher content is obtained, and this method sometimes affords relief from breathlessness The gas should be first led as a continuous stream of bubbles through warm water, a stream of dry gas is not tolerated In recent years special inhalers have been introduced, delivering high concentrations of oxygen, which often give notable relief Of these the B–L–B inhaler is recommended An oxygen chamber may be used

Cardiac tonics and stimulants —The most emphatic action of digitalis and its allies is in the case of auricular fibrillation, to be considered later Clear evidence that digitalis is useful in failure with congestion presenting normal rhythm has been slower in coming, because success in such cases is less obvious and less frequent Nevertheless it is now generally recognised that digitalis is valuable in these cases and sometimes succeeds in lowering venous pressure and in bringing relief where other remedies have failed Such successes are won with full doses of the drug initial doses equivalent to 2 drachms (3 5 c c) of tincture (1 5 mg digoxin) and further dosage at the rate of 1 drachm (1 7 c c) of tincture a day The underlying principles of digitalis administration are indicated on page 97, except that the height of venous pressure is watched rather than heart rate There is no evidence that digitalis, given in small doses (up to 20 minims or 0 5 c c of tincture daily), recognisably influences the progress of uncomplicated congestion, either at a late or at an early stage Its administration to heart cases, merely because they are heart cases, is to be discouraged

I have seen no patient respond to injections of strychnine or camphor Coramine, 1 to 2 c c intravenously, has a limited value Alcohol is sometimes useful, not as a stimulant, but in overcoming restlessness and sleeplessness

Pain over an engorged liver or over an enlarged heart is to be treated first locally by the ice bag, or by irritants, such as a mild capsicum plaster The application of six leeches to the hypochondrium will sometimes completely relieve when other remedies have failed, morphia is the final and the best remedy for precordial pain.

Cough —Where cough is the result of an associated bronchitis, the expectoration of stiff mucus is helped by alcohol and stimulants such as ammonium chloride (see page 261), where it is the result of commencing congestion and oedema sedatives (as for example codeine and heroin) are more suitable The object is in both cases *to decrease the effort of coughing*

Oedema drainage —In cardiac failure pitting confined to the feet or lower parts of the legs usually disappears within a few days of the beginning of bed treatment this may result simply from re distribution of the excess fluid it may result from rapid improve ment of the circulation In cases of early dropsy, no other remedies are required, though it is wise to restrict the fluid and salt intake In only a proportion of cases does dropsy call for special treatment for dropsy is an end result of cardiac failure and as failure is relieved dropsy disappears Treatment should be directed, therefore, first of all to the circulation but special remedies for dropsy should be started promptly in cases in which the accumulation of fluid is spreading to the trunk threatening the serous cavities, or in which congestion is responding little or slowly to treatment In such cases *while the fluid intake is reduced as closely as possible to 20 oz* (570 c c) and salt to about 15 grains (1 g) a day, the measure of urine passed daily should be watched and diuresis encouraged Diuretics of the xanthine series are among the most potent we possess Dimethyl xanthine (theophylline) is the favourite com pound (3 grains or 0 2 g thrice daily) aminophylline is a combina tion with ethylene diamine (same dose) and theocin is a combination with sodium acetate (dose 5 grains or 0 3 g) Diuresis comes within the first few days Gastrointestinal symptoms headache, and visual disturbance, sometimes prevent the drug s continuation Theo bromine an isomeric dimethyl xanthine (dose 10 grains or 0 6 g), diuretin, a combination of theobromine with sodium salicylate and caffeine (trimethyl xanthine) are less useful In recent years mercurials have been introduced as diuretics and some of these are very powerful Ammonium chloride (6 to 8 tablets of 10 grains or 0 6 g in 24 hours) given first for three days to induce acidosis tends to increase the effect The B P preparation mersalyl is injected as *a 10 per cent solution in 5 per cent dimethyl xanthine* of which 2 c c is injected intravenously or preferably intramuscularly in the morning great care being taken that it does not enter the sub cutaneous tissues, and the result observed the dose may be repeated as often as 10 or 20 times at intervals of two or more days This

diuretic usually gives a copious flow of urine (60 to 80 oz or 1700 to 2250 c c) It is a most valuable drug There are now several at least equally potent alternative mercurials There should be no repetition if a first dose is followed by substernal oppression, a tendency to faintness, or disturbed cardiac action Defects of renal function contraindicate the use of the drug

When dropsy of the legs has spread up the trunk, the patient should be kept in a sitting position, so that the fluid may collect in the dependent legs and not in the thorax and abdomen The patient sits by night and day well propped in a wide deep, and comfortable chair, or preferably, because it entails no lifting, the cardiac bed is converted into a chair (Fig 5) If in these circumstances the legs become very large drainage must be considered The number of cases treated by drainage should be small the chief indications for tapping are failure of other remedies to relieve conspicuous swelling especially increasing swelling, and dropsy of the trunk persisting in the sitting position In cardiac failure uncomplicated by renal disease I have rarely seen serious sepsis follow an inch long incision into the subcutaneous tissue of the dorsum of each foot, or the insertion of Southey's tubes With abundant sterile dressing both methods are safe, but as the fluid escape is freer with incisions, this method is to be preferred Drainage may be continued for days or a week or more, it is brought to an end because the dropsy has subsided, because the flow ceases and dropsy though decreased is stationary, and not infrequently because it begins to cause weakness of the patient

A serious difficulty in treating obdurate oedema is blistering and excoriation of the skin Blisters should be opened and all raw surfaces carefully guarded with a weak sulphanilamide paste It may become necessary to raise the legs above their usual dependent position to obtain healing, but it is done at the cost of moving the dropsy higher up

Tapping the chest in cardiac failure is more often required for unilateral than for bilateral accumulations, which can generally be displaced by posture and diuretics When aspirating, the needle should be inserted in the line of the scapula about the 8th inter space, whenever possible, the patient should be lying on the side Not more than 1 pint (500 to 600 c c) fluid should be withdrawn without oxygen replacement

Tapping the abdomen in cardiac failure is called for in those occasional cases of rheumatic heart disease in young people, in

which fluid collects and persists in the peritoneum rather than in the legs It may be repeated as it is required, and may be used in anasarca when the ascites is large

CASES WITH UNDUE BREATHLESSNESS (PULMONARY CONGESTION AND OEDEMA)

Undue breathlessness in cardiac failure may be persistent, associated with periodic breathing, or appearing in the form of attacks of cardiac asthma Patients of these types are often difficult to relieve Postural treatment should be invariable, whether orthopnoea is present or not A milk and vegetable diet is recommended Oxygen is occasionally effective Aminophylline and opium (see pages 70 and 40) are drugs that are serviceable in warding off attacks The former is also of much value in its relief of the distress of periodic breathing Patients presenting continuous evidence of congestion and oedema of the lungs are sometimes much relieved by mersalyl injections

In the severe attacks, whether signs of oedema are clear or not, morphia (¼ grain or 0 015 g) should be given at once, it is the most effective remedy known, and usually brings relief within a half to one hour Oxygen may be given Nitrites relieve mild attacks occasionally Digoxin or strophanthin injections have found advocates Adrenaline should on no account be given If the veins of the neck become turgid prompt bleeding is indicated

The treatment of acute pulmonary oedema is similar.

CASES PRESENTING AURICULAR FIBRILLATION

The general principles underlying the treatment of failure with congestion, and many of the details of treatment, are the same whether the auricles beat regularly or are fibrillating Most of the cases actually belong to the latter class, and in certain respects the presence of fibrillating auricles usually calls for modified treatment The work of recent times has shown that the chief service of digitalis is to control ventricular rate in cases of this type By slowing the ventricle it decreases the expenditure of energy, and by slowing it increases the length of diastole relative to that of systole This action of digitalis in conserving the heart's energy and prolonging its rest is in harmony with the chief principle of treatment of cardiac failure and justifies digitalis being called a cardiac hypnotic The action of digitalis in auricular fibrillation is so potent that in cases of failure with rapid ventricular action,

rest of the body in bed combined with slowing of the ventricle by digitalis usually suffices to ensure rapid disappearance of the signs of failure The response to digitalis in these cases can be looked for with so much confidence that venesection, unless congestion is con spicuous initially, can usually be avoided Special measures for the relief of oedema, the use of the stronger diuretics or actual drainage, should await the full response to digitalis, and they then usually prove to be unnecessary

Since digitalis benefits congestion in this class of case indirectly, and the treatment of auricular fibrillation by this drug is the same whether congestion is present or not, the details of this treatment will be given at a later stage (page 97)

AFTER CARE

Many cases of severe failure with congestion and most cases of less severe type, including those presenting fibrillation of the auricles respond well when treated along the lines previously in dicated The necessary treatment will naturally be less drastic from the beginning in the cases that were originally of mild type, and in the severer cases a rigid system of rest and other parts of the regime may be relaxed as recovery happens But there should be no thought of ending rest either in the severer or milder types of cases, when the signs of increased venous pressure disappear The heart has recovered no more than to regain its capacity to carry on a circulation adequate to the needs of the resting body, it still altogether lacks reserve This reserve becomes re established in greater or lesser degree by enforcing a further period of almost com plete bodily rest and the rate of recovery is slower in cases originally of the severe type No patient who has shown definite signs of venous congestion should leave his bed for several weeks after all these signs have vanished A total period of six to eight weeks in bed is rarely too long it is often too short, to ensure maximal benefit Patients who recover slowly, or imperfectly, must often lie quietly for three to six months or even longer Cases that are allowed up too soon quickly revert to their former state, haste in treatment does not save time Prolonged rest should be rendered less irksome by arranging mental occupation such as reading or being read to, wireless telegraphy, simple games or handwork, by permitting the patient to do more and more for himself as he recovers strength, by transferring him to a couch or chair for some hours each day, by employing massage, especially to the legs, and later passive

and gently resisted movements, and by an early return to fuller dietary The steps taken towards increased liberty of action are to be small and not too frequent At no stage of treatment should tobacco be allowed

Return to exercise, supervision —The guide to a return to bodily activity is the response to effort A patient has recovered normal venous pressure and has been for a further period of several weeks confined to bed, at the end of this time he washes himself, sits up, and lies down without a trace of breathlessness Such a patient may be permitted to walk across his room to a couch and, after some days, to walk a few minutes up and down the room A schedule of increasing exercise cannot be laid down beforehand, but for weeks nothing must be done that gives a noticeable increase of respiratory rate or fatigue Walking in the room may be replaced by short walks on the flat out of doors, the bed being arranged downstairs, or the patient carried up and down the stairs each day, for the latter purpose a special carrying chair having two large wheels and long handles fore and aft is useful As tolerance of exercise improves, exercise may be extended, until perhaps a few miles are walked on the flat at a moderate pace Few patients who have experienced severe failure will advance beyond this point, the milder cases will, and should be encouraged to do so However much the most favourable case improves, it is never good practice to allow these patients to call forth their full reserves or to press exercise to the point of bodily discomfort Strenuous acts (page 291) should be forbidden for all time The patient should adopt strictly healthy habits (page 289), he should report himself at frequent intervals, or at once if he feels unwell

The hours in bed should be longer than is customary, and any tendency to fatigue may be counteracted by the patient lying down for a suitable period during the afternoon.

A diet suited to a convalescent adult, still at rest in bed or chair, is detailed later (Diet II, page 292) As convalescence becomes more real, and bodily work is undertaken, more food is needed

REFRACTORY CASES

The rule that confines patients to bed until after all signs of venous congestion have vanished has sometimes to be broken The instance may be that of a patient already treated several times for congestion, and responding very slowly to treatment In the

latest illness prolonged treatment brings the patient to an improved condition but one in which the veins and liver remain definitely engorged the condition is stationary for perhaps another three or four months and the patient becomes restless under his confinement These are very serious cases but some increased liberty of action has often to be allowed them

Thyroidectomy (total) has a limited value in such cases reducing as it does the energy expenditure of the heart It should be used only in selected cases and confined chiefly to those presenting high basal metabolic rate

CHAPTER VI

SKIN COLOUR AND CIRCULATORY RATE

Pallor

The blood coloration of the skin is due mainly to blood in the networks of capillary venules in the subpapillary layers of the dermis The cause of pallor is reduction in the amount of haemoglobin held within these vessels, and this happens when the haemoglobin content of the blood is low or when the amount of blood held in the vessels is decreased

In the heart disease of adults persistent and conspicuous pallor, especially when the skin has a yellowish or subicteric tinge, is almost always due to streptococcal infection of the heart valves Conspicuous pallor in children is often the result of active rheumatic infection These pallors naturally involve both the skin and mucous membranes, for they are associated with actual anaemia of the secondary or hypochromic type Heart disease is occasionally accompanied by secondary anaemia caused by local infection or arising from haemorrhage These are the main associations of heart disease and actual anaemia Pallor in cases of heart disease should never be dismissed lightly, it is an invaluable guide to infection Dilatation of the heart in pernicious anaemia may be mistaken for heart disease with infection

A bloodless condition of the skin is not infrequent in the advanced mitral stenosis of young girls, it is also frequent in the terminal phase of failure with congestion, in these it does not involve the mucous membranes, and is evidently due, not to general anaemia, but to actual emptiness of the minute cutaneous vessels

Transient pallor occurs in two sets of circumstances Firstly, when there is a simple drainage of blood from the skin in syncope (page 107), such pallor may be intense, it is seen especially in the face of the standing or sitting subject, and is inconspicuous in dependent parts Secondly, it occurs in the rare attacks of vaso

constriction described on page 72. In these the skin is affected universally and the hands and feet become pale although they lie below the level of the heart

Cyanosis and its Interpretation

CAUSE OF CYANOSIS

The cause of cyanosis is an increase in the amount of reduced haemoglobin, in any form, within the small vessels of the skin or mucous membrane These reduced pigments appear blue through the skin The condition may result when the blood pigment as it is brought to the integument, in which cyanosis is observed, is less oxygenated than is normal haemoglobin, or it may be due to an unusual loss of oxygen by the blood in its actual passage through the skin or mucous membrane In some instances each of these two factors is in part responsible We shall consider the pure examples first

Cyanosis arising centrally —In this variety of cyanosis, the blood as it leaves the heart contains haemoglobin in forms other than oxyhaemoglobin It occurs in any condition in which air is prevented from reaching the alveoli in sufficient quantity, but these are often impure forms of cyanosis, being associated with cardiac failure, and are dealt with later The most familiar and striking example of a condition of pure central cyanosis is that of congenital heart disease, in which the main lesion is a gross septal defect, allowing a quantity of venous blood to pass freely from the right heart into the aorta, thus short circuiting the lungs and remaining unaerated Another example is that in which methhaemoglobinaemia or sulphhaemoglobinaemia occurs Pure examples of cyanosis arising centrally are usually easy to recognise Such cyanosis is often first suspected because it is conspicuous, because, although conspicuous, it is accompanied by little or no breathlessness while the subject is at rest, or because it is unassociated with signs of venous congestion It is a general cyanosis, affecting not only the skin of face and hands, but the mucous membranes even within the mouth and the skin of the trunk, the last, however, often contains too little blood to yield an obviously cyanotic tint A more important statement than any that precedes, because applying exclusively to this form of cyanosis, is that it occurs in superficial skin and mucous membrane that is spontaneously warm or hot Warmth or heat implies a rapid flow

of blood through the skin that displays it, and cyanosis of central origin remains, however fast the blood travels through the skin

In the form of cyanosis considered, the skin in which cyanosis is observed is usually warm, but if it is suspected in a skin that is cool or cold, it is my habit to provoke an increased flow of blood Thus, if the lobe of the ear is deeply cyanotic and cool it can usually be made warm by local massage, preferably it should not only be come warm but should display capillary pulsation (page 51) after the massage Where cyanosis is central in origin the ear lobe remains cyanotic even though it becomes hot it will become a little brighter in colour if the cyanosis in part originates centrally and in part peripherally Another method is thoroughly to warm a cyanotic hand in water at 35° C (95° F) then to stop its circulation for seven minutes, and finally to observe the tint of the reactive hyper aemia (or flush) that pervades the skin when the bandage is re leased Normally and in cases of peripheral cyanosis presently to be considered, the colour of the quickly appearing flush has the bright redness of arterial blood but where cyanosis is of central origin the flush appears bluer than normal By using the signs and tests described cyanosis that is purely central can always be recog nised at the bedside These methods lead up to the diagnosis of many cases of congenital heart disease or of more occasional ab normality of blood pigment An examination of the blood pigments spectroscopically differentiates between the former and the latter, so does an examination of the heart when this uncovers clear signs of congenital disease

Cyanosis arising peripherally—This variety happens when the rate of flow through the skin diminishes sufficiently In its slow transit the blood is robbed by the cutaneous tissues of an unusually high percentage of its oxygen On the one hand, slowness of flow may be confined to superficial tissues like the skin on the other hand it may result from failure of the general circulation We shall consider the former instance first Slowness of blood flow mainly affecting the skin is usually a response to low surface temperature In interpreting cyanosis in various conditions, the temperature of the skin in which it is occurring is of very great importance Cyanosis is generally noticed in the regions of skin that are most highly coloured, namely, in the face and hands and these are just the parts that vary conspicuously in temperature in response to changed environment It is familiar knowledge that in cold weather these parts of the body frequently become cyanotic in people otherwise

regarded as normal, slight but distinct cyanosis of the skin is in fact a normal accompaniment of skin temperature between 15° and 25° C (59° and 77° F), and in many subjects this discoloration is exaggerated The cyanosis is due to diminished rate of flow, and this in turn is due to constriction of the arterioles of the skin in response to the low temperature Cardiac patients are just as prone as normal people, if they are not more prone, to display this purely local change The unreliability of cyanosis as an evidence of general circulatory efficiency arises largely out of this fact and out of the difficulty of distinguishing in the skin between slowness of blood flow that is local and that is general, there is no constant relation between the blood flow to superficial and deep structures

Cyanosis of failure with congestion —It is erroneous to believe that cyanosis in cardiac failure results simply from deficient aeration of the blood in the lungs this is probably never the case It is true that in advanced failure and very deep cyanosis the blood may leave the aorta in an imperfectly oxygenated condition, consequent upon its passing through alveoli that are airless or relatively so owing to congestion or oedema of the lungs But in the same cases the flow of blood through the skin is slow, and the visible cyanosis thus receives a peripheral contribution which is substantial In states of lighter cyanosis the peripheral factor is the chief or the only one concerned Cyanosis in cases of failure with congestion is found in skin or mucous membrane that for the most part is cool or cold

When, in a case of failure with congestion, cyanosis is found in a skin that is cool or cold, it is not very useful to try and ascertain if this cyanosis arises in part centrally It would be of greater interest to ascertain whether the blood flow has become slow locally in response to coldness of the skin or whether the slow circulation is primary and the cold skin has followed as an effect In the latter case, when the skin cools its arterioles likewise respond by contraction to the lowered temperature, and the cyanosis tends to be a little deeper for a given level of temperature than in the former case But tint and temperature cannot be gauged by the unaided senses with nicety, and there are wide individual variations and so it is frequently found to be impossible to decide clinically between these alternatives only one of which signifies a cardiac defect In cardiac cases the evaluation of minor grades of cyanosis in facial or digital skin that is in fact cool or cold is difficult

Though recognition that a patient seen for the first time has cyanosis may provide the earliest hint of cardiac disability, it will

be apparent from this discussion that it is quite precarious to judge the general state of the circulation from this phenomenon To use cyanosis when present as a sign of systemic venous congestion is to blunder into accepting indirect and imperfect evidence, when the direct evidence in the veins, described in an earlier chapter is available Cardiac patients may develop cyanosis of central origin and yet display little systemic venous congestion this is specially the case in bronchitis (page 259) and in oedema of the lungs (page 20) For similar reasons it is unwise to draw conclusions as to the general condition of the circulation from the absence of material cyanosis Many patients, undoubtedly and severely congested, present *very little obvious cyanosis* *Firstly, this may be due to simultaneous* pallor of the skin, for the appearance of cyanosis is very dependent upon the presence of a full amount of blood pigment in the skin Relative emptiness of the small vessels that give the skin colour is frequent in failure with congestion and thus the tint of the blood is concealed similarly it is hidden when, as is not infrequent, actual anaemia is present On the contrary, full vessels or a polycythaemic state emphasise a deficiency in the aeration of the blood as this is judged by skin tint Secondly, it may be due to relative freedom of the lungs from congestion

MALAR FLUSH

A patch of colour of unusual depth over the region of the malar process has been thought to be a sign of mitral stenosis It is not related causally to this disease In very many young people who have been exposed frequently to cold wind, a bright malar flush is present, in many others the vessels in this region, though less conspicuous, possess less tone than do those of other parts of the face. The skin of this region of the face is therefore particularly prone to be involved in flushes of any kind, among other causes venous congestion brings this region into prominence, rendering it bright and a little cyanotic The malar flush comes to be associated frequently with mitral stenosis, because in youth failure with congestion is so often accompanied by this valve lesion The only value of the sign is in directing attention to the veins

CUTANEOUS BLOOD FLOW

Cyanosis is a reliable sign of slow blood flow through the skin provided that the entering blood is fully arterial Actually a distinct

cyanosis in these circumstances indicates a very greatly reduced flow And yet the flow may be very slow and the skin bright red, this is so when the skin is unusually cold (below 10° or 15° C, 50° or 59° F), the reason is that at low temperatures the blood can give up little of its oxygen If the skin of the hand is blanched by pressure and the blood returns slowly, it is clear that rate of blood flow in the skin is slow, but a rapid return cannot be interpreted in the reverse sense, since it happens when the blood flow to the limb is arrested provided that the skin is already turgid with blood Whether the skin is cyanosed or not, the depth of its colour is an uncertain gauge of the amount of blood flowing through it, in general the pale and not the flushed areas of the facial skin have the greatest flow of blood through them The best gauge of blood flow in the skin is not colour, but temperature Spontaneous warmth of the skin shows it to be possessed of good circulation, irrespective of tint or depth of colour

Capillary pulsation, so called, or visible flushing and paling of the skin with each heart beat, is a sign of locally dilated arterioles It can be induced in any young and normal subject by heating the

high pulse pressure, but chiefly to the vasodilatation commonly prevailing in this condition (page 137) It occurs only in warm or hot skin or mucous membrane It has no clinical value apart from the evidence it provides for the state of the arterioles, consequently it should not be elicited by rubbing or warming the skin, but should be looked for as a spontaneous phenomenon as such it is much more frequent than has been thought It occurs less frequently under the nail than in the pulp of the finger and in the facial skin

CHAPTER VII

CARDIAC ISCHAEMIA CORONARY OCCLUSION

Coronary Arteries and Cardiac Ischaemia

THE CORONARY ARTERIES

There are usually but two arteries, the right and left coronary, supplying blood to the heart, these lead off directly from the aortic wall and, although anastomosing in a limited way with each other, they anastomose scarcely at all with other arteries in the body The coronary territory is an almost isolated territory, and such connections with other vascular territories as exist are minute and occur in the parietal pericardium and in the walls of the main vessels The heart is really dependent upon two arteries for its supply, and obstruction of these is an event for which there can be little compensation by collateral circulation It is difficult to believe, though there are some that do, that the muscular walls may be nourished by a backward stream through Thebesian veins The two coronary arteries must supply the heart, whatever the amount of blood it needs They must supply not only the normal sized, but also the greatly hypertrophied, organ, they must supply the muscle adequately, not only when its work is suited to the needs of the resting body, but when the heart is called upon, as it often is, to do a greatly increased quantity of work

Pathological anatomy —Disease interfering with the blood supply of the ventricular muscle occurs either in the length of the coronary vessels, or at their mouths In the first instance the disease is a part of an atheromatous change in the vessels of the body generally, though the coronary vessels may be affected most severely, the narrowing results from gradual thickening of the vessel wall Obstruction at their mouths is usually due to disease of the aorta itself, and this disease is atheromatous or, more commonly, the fibrosis of syphilitic aortitis

Coronary vessels are also liable to sudden obstruction Thus, on occasion a free embolus or the end of an aortic valve vegetation is driven into an orifice and blocks its mouth A much more frequent source of sudden obstruction is thrombosis, this occurs in the length of the artery, and only in such as are already diseased The changes in the muscle consequent upon coronary occlusion are described on page 55. Coronary arterial disease is often accompanied by diffuse fatty degeneration or diffuse fibrosis of the ventricles (see page 254)

ANGINAL PAIN AND MUSCULAR ISCHAEMIA

It is known that when a coronary artery is acutely obstructed, pain usually results, this pain is anginal in character and distribution Pain of similar character is produced in the muscles of a limb that are worked when a tourniquet stops the flow of blood to them It is probable that anginal pain is caused by a specific chemical or physicochemical change *in the musculature of the heart, and that this is* usually, if not always, consequent upon defective blood supply Almost all, if not all, the phenomena relating to anginal pain are explained upon the basis of this theory, many of the phenomena have been explained as yet in no other way While it is desirable to eliminate theory from this book as far as possible, the conception that muscular ischaemia, absolute or relative, is the cause of anginal pain, so simplifies an understanding of the circumstances in which this pain comes and goes, that it is advantageous to model description around this idea When a coronary artery is blocked, a corresponding area of muscle loses its blood supply, there is a state of absolute ischaemia When the supply to an area of muscle is reduced, or when it is incapable of increasing sufficiently to bring an adequate supply to muscle called upon to do an unusual amount of work, there is a state of relative ischaemia That the first state gives rise to anginal pain is certain, that the second does is unproved, but highly probable, especially from recently accumulated evidence Theoretically, pain will occur when relative ischaemia affects the ventricular muscle as a whole or, more usually, in part To be complete, the conception of ischaemia includes, not only change in the structure of the coronary vessel, but the state of tone of coronary artery and arteriole, it allows that a deficient supply may be caused or contributed to by undue coronary tone, and that a deficiency may be made good by coronary vasodilatation

The long lasting effort to form a simple but complete classifica

tion of patients who complain of anginal pain is not even yet fully successful Our ideas of angina have changed in late years because the symptoms of coronary thrombosis have been recognised and because this malady is no longer confused with allied maladies There remains a number of comparatively rare and diverse types that are improperly understood The chief types only will be described in this and the next chapter

Coronary Occlusion with Infarction

The symptoms and signs of coronary thrombosis and embolism are alike and a description of thrombosis will adequately illustrate the occlusion accident

Thrombosis of a coronary vessel is an accident occurring chiefly in men in their fifties or sixties it happens occasionally in women it is rare before the age of forty The subjects usually present signs of arterial disease and more often than not high blood pressure Coronary thrombosis is comparatively rare in cases of syphilitic aortitis Although it not infrequently happens in patients who have already presented or are showing signs of failure it is rare in rheumatic heart disease and very rarely occurs in cases of auricular fibrillation A considerable number of those who suffer have complained of earlier anginal symptoms but some are affected without warning while in robust health The malady is not infrequent in father and son or in two brothers

PATHOLOGY

The cause of coronary thrombosis is to be found in disease of the vessel affected such disease is usually but a part of a general arteriosclerosis Thromboangeitis is a rare cause Trauma is I think occasionally and directly responsible No coronary vessel is exempt from thrombosis but the descending branch of the left coronary is affected more frequently than any other probably because of its relatively exposed position The vessels next but less prone to be affected are the circumflex part of the left coronary or the last part of the right coronary artery The coronary arteries anastomose though not very freely with each other If such a vessel as the descending branch is tied in a dog a part of the ventricular wall loses its blood supply becomes dark in colour and bulges with each systole of the heart In many instances and within a few minutes of ligation the ventricle may begin to beat irregularly owing to the occurrence of ventricular extrasystoles single or grouped and in a

number fibrillation of the ventricle comes and at once kills the animal In others the heart continues to beat, though less efficiently, owing to part of its wall being thrown out of action In a few instances, where the territory is small, the circulation is re-established through collateral coronary channels and the action of the muscle becomes restored

In the human subject similar events undoubtedly happen When the blood supply to a part of the ventricle is permanently arrested and the subject survives, an area of muscle, probably smaller than that originally supplied, becomes infarcted and undergoes a fatty necrosis or "myomalacia cordis", becomes soft and fragile, and may rupture Ultimately the necrosed tissue is replaced by scar tissue, which is firm in about six weeks The position and size of the muscle area involved naturally depends chiefly upon the position and size of the vessel that is blocked In the usual instance of thrombosis of the descending branch the area affected is mainly in the front and apical regions of the left ventricle, and is from 3 to 6 cm in diameter, it involves a corresponding area of septum The wall of the heart is always more affected on its endocardial than upon its pericardial surface, the latter often escapes, upon the former it is usual for blood clot to form sometimes in an extensive and deep mass The necrosis of the wall the endocardial thrombosis, and involvement of the pericardium, are each responsible, as we shall see later for special symptoms or signs

Similar changes may occur when the blood supply is greatly reduced without actual occlusion

SYMPTOMS AND SIGNS

Coronary thrombosis usually occurs when the subject is at rest, sitting in a chair or lying in bed, or actually during sleep, it also occurs at other times The symptoms may be so slight and few that the patient seeks no advice, they may be distinct or severe The usual symptoms and signs may be divided into those occurring as immediate effects, which include sudden death, pain, and the manifestations of collapse and cardiac failure, and into those that are delayed

Death —In a considerable proportion of the cases death occurs very rapidly, there may not even be time for a complaint of pain Many subjects are found dead These sudden deaths are due in all probability to fibrillation of the ventricles

Pain and collapse —Pain is not invariable When the pain comes

it may be slight and may remain so, more often it rises rapidly to its culminating point and usually from then on is severe, often agonising it is a continuous pain in the sense that it is unfluctuating It is gripping or aching in quality, generally it starts over the sternum, it may remain there or it becomes more diffuse as it strengthens and spreads to involve the left or right or both arms, but especially the left, or to the neck and jaw, or to the interscapular region, or to the abdomen The lower sternum or the epigastrium is more frequently the starting point than in angina of effort During the period of pain the patient may rest immobile, but frequently shows agitation and is restless or rolling about in agony, unlike the pain in the angina of effort, movement does not seem to increase it appreciably Though its duration varies, the pain is characteristically long lasting, a feature which renders the malady so distressing It lasts usually for one or more hours at its height, and declines gradu ally over a long period of many hours or several days

The pain is usually accompanied by a state bordering on collapse, the patient being pale, haggard, anxious, weak, and often sweating profusely When this pallor is associated with cyanosis the face assumes a light blue grey colour that has been thought character istic of the malady In this state of collapse nausea is frequent, and vomiting and movement of the bowel may occur The pulse is small or it may be impalpable the blood pressure is lowered, and much lowered in cases where it was originally high, the urine is scanty The heart rate is usually increased but may be normal or slow in rate This group of symptoms often resembles closely that of vasovagal attacks (page 109), though with quickened pulse the re semblance becomes less distinct

Manifestations of cardiac failure —Symptoms and signs of car diac embarrassment are frequent but by no means invariable, their degree naturally varies with the extent of myocardial infarction and the state of the remaining muscle, breathlessness is almost invari able, often severe, and sometimes it is the only prominent symptom Cyanosis is common Signs of venous congestion may be clear or relatively inconspicuous They may appear quickly in any degree or may increase gradually The heart sounds are indistinct a systolic murmur may appear in the apical region, the heart may exhibit extrasystoles, paroxysmal tachycardia, or auricular fibrilla tion, the last two, if present, will increase the circulatory embarrass ment When the upper part of the ventricular septum is involved, heart block may appear Electrocardiograms generally present

changes of a peculiar and characteristic kind (Fig 6) The signs in the heart and in the veins are usually far less than would be expected from the suffering of the patient Periodic breathing and gallop rhythm are common Signs of pulmonary congestion and oedema often predominate notably over those of systemic venous congestion, breathlessness may predominate, there is persistent cough with scanty frothy and sticky sputum, in severe oedema the sputum is serous and bloodstained, and rales and rhonchi appear over the lungs, sometimes this oedema develops very rapidly and kills the patient

Delayed signs (fever, leucocytosis ruptured heart embolism, and friction)—As is usual when a portion of a parenchymatous organ

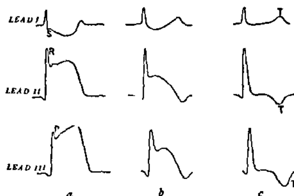


FIG 6—A diagram to illustrate one of the forms of electrocardiogram found in cases of thrombosis of a coronary artery The curves shown in column *a* are seen in cases of fresh infarction of the posterior wall of the heart, the characteristic feature is the plateau form They change to those of columns *b* and *c* within a few days or weeks the plateau becomes less conspicuous and inversion of the end deflection *T* occurs in one or more leads In anterior infarction the curves are in general similar if we transpose leads *I* and *III*

loses its blood supply and dies there is a febrile reaction This fever is low, amounting to 1 or 2° F The temperature rises usually on the second day of the illness it may rise earlier, it continues for a number of days and occasionally for as much as two or three weeks there may be no fever Associated with the death of the muscle and its d

a

a

millimetre

The weakness of the ventricular wall, which is at its height during the second week after the accident, is responsible for the special

accident of rupture of the wall, haemopericardium, and rapid death The accident is rare before the fifth day and after five weeks

As has been stated, clot usually forms on the affected portions of endocardium, and this clot may become detached in small or large pieces and give embolism The emboli may enter and plug any artery in the body, but are more common in the systemic than in the pulmonary system Embolic accidents give rise to numerous complications, for example, hemiplegia, loss of circulation to a limb, splenic infarction and so forth, the presence of albumin or red blood cells in the urine consequent upon small renal emboli is often valuable for diagnostic purposes

When the pericardium is involved in the infarction and the area is on the front of the heart, friction sounds appear over the central and lower portions of the precordium This pericarditis usually appears on the second or third day of illness and lasts a few days Occurring in perhaps a third of the patients, it is a most important diagnostic sign and therefore should be sought for closely and daily where the diagnosis is in doubt

DIAGNOSIS

The picture in its typical form is too characteristic to be missed by those who know of it The central feature is pain of anginal type that continues long beyond the expected time and fails to yield to nitrites These cases used to be diagnosed "status anginosus" When the pain is over the lower sternum or in epigastrium the trouble is apt to be mistaken for acute abdominal states especially for perforated ulcer or cholecystitis tenderness and rigidity of the upper abdomen and signs of collapse with vomiting occur in all The age and sex of the patient will often be helpful in differentiating for ulcer commonly occurs in the young and cholecystitis in women a previous history recognisable as clearly anginal on the one hand or gastric or biliary on the other is often to be elicited In thrombosis the patient will often speak of pain as present over the middle or upper sternum also Abdominal cases though pale are not cyanosed as are those of thrombosis, neither do they present the evidences of oedema of lungs or early signs of congested veins, either of which like the breathlessness accompanying each, may be of great value in differentiating The severity of the pain may be responsible for a diagnosis of gastric crisis a mistake which should not occur, because in this the pain is spasmodic and not continuous

When pain is less severe or mild, acute indigestion is apt to be

diagnosed and the true state may not become manifest until em bolism or heart rupture occurs or until angina develops shortly or perhaps after several years

If pain is mild or absent and breathlessness is the chief feature the diagnosis made is of an attack of cardiac asthma (page 21) acute cardiac failure of unknown origin or an anomalous form of pneumonia and the fact that brief and even severe pain has been present will not always prevent the first and last diagnosis Success in diagnosis will depend mainly upon having the possibility of thrombosis in mind and in watching for the delayed signs of the disease especially for the subdued fever the leucocytosis red cells in the urine and pericardial friction Cases of massive pulmonary embolism give symptoms that are similar even including electro cardiograms but pain is more often axillary than sternal and cyanosis and respiratory distress are more pronounced The sudden onset of symptoms or signs of cardiac failure in a middle aged patient unexposed to heavy work and afebrile at the time should always arouse suspicion of coronary thrombosis When there is coronary thrombosis but the diagnosis is in doubt electrocardio graphic examination will often resolve the doubt (Fig 6) but patients should not be taken to the instrument

COURSE AND PROGNOSIS

The course of coronary thrombosis is very variable The mortality in the stage of the acute and severe illness was at first greatly over estimated Undoubtedly in a number death occurs at the onset or abruptly after a day or two and presumably from ventricular fibrillation others succumb to rapidly deepening failure of the sys temic circulation or are suffocated by a rapidly developing oedema of the lungs some are killed by rupture of the heart others by rarer accidents such as the syncope of complete heart block or gross embolism of brain or limb

Those that recover may be severely crippled in good health or in any intermediate state the ultimate state depending largely on the condition of the remaining heart muscle and of the vessels supplying it Patients previously suffering from heart failure or from angina who survive are usually severely damaged failure is increased congestion becomes established and progresses with varying fluctuations in intensity slowly but surely to a termina tion Anginal cases are in general less tolerant subsequently of exercise Curiously however some such patients lose their

pain after thrombosis a possible explanation is that the original anginal pain arose from an area of muscle supplied by a narrowed artery, that this vessel becomes thrombosed, and that the pain giving muscle then dies Many of the survivors are left with very limited capacity for exercise owing to anginal pain This may be a remnant of the past, or may appear during convalescence or at any subsequent time Many patients are known to recover to good health leading active lives or relatively active lives The prognosis in these is uncertain owing to the development of angina, and owing to recurrence of coronary thrombosis in about one case in four Originally regarded as a hopeless malady, further observation has much lightened the gloom of the older outlook It has become recognised, as diagnosis has increased in accuracy and from the finding of scars of infarction in subjects unsuspected of the disease that minor attacks of thrombosis are frequent in the middle aged and elderly, and that such attacks often leave the patients seemingly unchanged It has become known too that of those who convalesce well even from serious attacks some return to active lives for periods of five ten fifteen, or more years others live on in comparative activity for periods up to fifteen or even twenty years

TREATMENT

The prominent symptom is pain and for this morphia is the best remedy the doses often need to be heavy (½ grain or 0 03 g , repeated in a few hours) Morphia is also useful in inducing the rest that is so desirable Even morphia however, frequently fails to give relief Opium is sometimes more successful (1 to 3 grains or 0 06 to 0 2 g) In cases of very severe continuous pain that is unrelieved by opiates light anaesthesia may be induced Nitrites are usually without effect but they should be tried, and so should aminophylline up to 5 or 10 grains (0 3 to 0 6 g) doses The patient is nursed in bed as is a case of grave cardiac failure Rest in bed should be continued for from six to eight weeks to ensure firm cicatrisation of the ventricular wall, during the whole of this period the patient is to be guarded by day and night nursing and helped in every way to avoid voluntary movement or effort Patients have lost their lives and especially those who have early recovered from symptoms by neglect of these precautions The after care is similar to that of cardiac failure or of angina with comparable symptoms

CHAPTER VIII

ANGINA PECTORIS

Angina of Effort

The malady originally described by Heberden under the term 'angina pectoris' is one in which pain of characteristic type comes during effort It is by far the commonest form of malady in which anginal pain occurs The pain may be interpreted as resulting when the blood supply to the heart, or part of the heart, is limited and consequently inadequate when the heart is called upon to expend much energy it is a condition of relative ischaemia

CLINICAL ASSOCIATIONS

It is essentially a malady of middle aged or elderly men, though it occurs exceptionally in women of the same age period and occasion ally in younger males It is commonest in sedentary and well fed people, general arterial disease is frequent and about a third of the males and most of the females display high blood pressure Disease of the coronary vessels either narrowing of their mouths or thickening of their walls with narrowing of lumen and old standing oblitera tion of a vessel are usually demonstrable after death A greatly enlarged heart, valvular disease especially aortic disease, may be found, but none of these is the rule A large fraction of the cases show no physical signs in the heart and many present only a few distinctly abnormal signs such as those of slight but definite cardiac enlargement, gallop rhythm, bundle branch block, and pulsus alternans Some of the patients suffer from cardiac asthma The malady is rarely associated with mitral stenosis It very rarely occurs in cases of fibrillation of the auricles or of failure with congestion

SYMPTOMS

Angina of effort not infrequently follows, immediately or later, upon an illness attributable to coronary thrombosis Usually, how

ever, this is not the case, and the first complaint is of discomfort or of actual pain on effort This is felt first of all, and in mild cases, on undertaking some accustomed act, such as running, or walking briskly uphill The sensation appears usually over the middle or upper part of the sternum, though it may be over lower sternum, over precordium, or more generally over the front of the chest It consists of a feeling of pressure, of gripping or of aching, it is smooth or continuous in the sense that it does not fluctuate in intensity from moment to moment or throb, though there may be slow changes in its intensity When high in the chest and at the base of the neck it gives a sense of strangling or suffocation, this originally gave the malady its name "angina ' If the subject stands still, the discomfort or pain goes away in a minute or two Many of these cases suffer very little pain because they become still at the first warning of its appearance More often than not there is no radiation If the subject presses forward the sensation usually grows in intensity, and may become more diffuse, spreading often to neck and jaw, *to one or other arm, to the abdomen or scapula* In the arm, and it is usually the left, the pain generally follows the inner aspect of the limb, descending to the elbow or rarely as low as the little finger Numbness in the fingers or arm is often experienced at the time or subsequently In rare cases the pain starts in jaw or arm and spreads from this to the chest Some patients speak of being able, by continuing exercise, to rid themselves of pain, but this is *uncommon*

In severer cases the same discomfort and pain is felt but is elicited by smaller amounts of work, as by walking briskly or at a moderate pace on the flat, the pain when it comes is apt to become more severe and to subside more slowly In these and the milder cases the amount of work necessary to induce pain varies little, sometimes astonishingly little as when a man walks out of his house and is brought *to his first standstill day after day at the same place*

In the severest cases pain is provoked by very little exercise, often rises to agonising intensity, and is repeated at relatively short intervals [illegible] for movement increases [illegible], ressed in his [illegible] d pressure is little raised, the pulse rate is as usual or is a little increased or decreased *Salivation may be free These attacks last a few minutes* or, recurring quickly, are drawn out for a period of a half an hour or more They leave the patient weak and tremulous, and the skin

of the chest, the intercostal muscles, pectorals, and sternomastoid often tender to touch

In attacks of pain produced by exercise, it is common to hear that they occur more easily in cold air than in warm, the reason is often to be found in the brisker movements of the patient in the former circumstance, in others it is suspected that cold reflexly contracts the coronary arteries The excitement of animated conversation, or anger provoked by dispute, is an adequate stimulus In some cases exercise is particularly prone to induce attacks when it is taken directly after food, this and the frequence of flatulence in the same cases may lead diagnosis to the stomach rather than to the heart

Some writers distinguish between the pain and the sense of tension or gripping in the chest, I am not satisfied that they are separate phenomena Few patients speak spontaneously of both, and many state that the pain consists of a sense of violent pressure or tightness, or that a sense of tightness grows until it becomes a severe suffocating pain, many others will not allow that gripping occurs

Expectation of death in the severer attacks is not often referred to unless the question is raised Such a fear is a common association of many new, painful, or disturbing experiences and is usually irrational There is no reliable evidence that a given sensation is responsible for it, or that it is more than an interpretation of a very unpleasant and new sensation or of an intolerable pain, it has at present little value in diagnosis Much depends upon the mentality of the patient, and something upon what he has read or heard previously about his ailment

DIAGNOSIS

The diagnosis of angina of effort turns, in subjects of suitable age and sex, almost exclusively upon the history, this should be elicited with the greatest care and will often require close and prolonged questioning Though pain may appear first in one of many places a history of pain over the sternum has more value than any other It is a serious error to imagine that anginal pain must be severe and that slight pain occurring on effort cannot possess grave significance In enquiring as to the type of pain, a most important point to elicit clearly is that the pain is uninterrupted, anginal pain does not throb or stab Sometimes it is important that final answers should be deferred until in the next attack, the patient has paid special heed to the particular points raised An extremely important matter is consistency in the history of the response to effort It is the rule that there is a given tolerance of work, constant within narrow

limits Patients with this malady do not relate that on one day the attack is brought about only after walking briskly, and that on the next it occurs spontaneously while the patient is resting quietly and undisturbed Single inconsistencies of this kind in the history may be explained occasionally by differences in external tempera ture, excitement or fulness of stomach but when inconsistencies are obvious and frequent the case is rarely one of angina of effort In angina of effort the pain of a given grade recurs with repetition of a particular act Isolated severe attacks of pain are not character istic of this malady but more of coronary thrombosis or of acute gastric affections

Minor inconsistencies are introduced when the attack is associated with large changes of pulse rate and blood pressure a different type described in the next section and major and frequent inconsistencies occur in cases in which the fault is not primarily in the heart

The presence of signs of definite heart disease will help to establish the diagnosis in cases of doubt but it should be understood that in many cases gross evidences are not forthcoming From this stand point some physical signs that help the identification of cardiac pain and which sometimes stand alone are described in Chapter XXV under myocardial involvement the most important of these are —gallop rhythm pulsus alternans and gross disturbance of the ventricular electrocardiogram such as bundle branch block and *inversion of T in leads I and II*

Relief by nitrites is very suggestive but is not diagnostic of the malady

Segmental pain —The pain of angina has a reference mainly corresponding to segments C7 C8 D1 and D2 it may extend out side these It is a general rule that pain can be referred from any deep lying structure supplied by nerves of the corresponding segment Thus it happens that pain of precisely anginal character and distribution may come not only from the heart but from [illegible]

upon the circumstance in which it arises I have on occasion realised that pain closely resembling angina originated not in the heart but in a muscle or aponeuroses of the chest wall and abolished it by local anaesthesia Pain of such origin may even come on exercise especially when the left arm is used In doubtful angina it repays to search the chest wall for area of tenderness and finding it to

anaesthetise it with novocaine injections

For parallel reasons when pain is conspicuous in lower thoracic segments than those named its possible origin from the gall bladder should be considered Such pain is also at times related to exercise and may find relief from nitrites

Angina of Rest

Anginal pain in resting subjects is usually an indication of grave disease Thrombosis of a coronary vessel may be responsible for a single severe attack a second thrombosis may cause a similar attack In patients who suffer from angina of effort and in whom the disease progresses far enough pain may be aroused eventually by such small stimuli that the patient takes to bed and still suffers attacks of pain

The commonest type of grave spasmodic angina met with in bedridden patients is different and has certain special features which make its separate description desirable The cases are usually young or middle aged male subjects suffering from aortic regurgitation females also suffer and the same syndrome is also found apart from aortic disease The aortic disease may be rheumatic or syphilitic The heart is greatly enlarged The coronary vessels may

best they are capable of only slight exertion for attacks of pain are easily induced by walking They experience severe attacks of pain while resting in bed This pain is of the usual type and has the usual and varying location and distribution of anginal pain It may or may not be associated with a feeling of intense anxiety The attacks may occur at any time but are especially prone to occur in the early hours of morning often waking the patient from sleep In the day they may be provoked by food or by the thought of food They last a few minutes to half an hour or more The patients usually state that the pain is accompanied by violent throbbing of

head The fac

sweating is of

neither cyanosis nor rale in the chest The heart rate is raised to 100 or 140 beats per minute the systolic blood pressure is raised from normal limits to very high points usually to 200 sometimes to 250 mm Hg or more these phenomena distinguish the attack

The pain, usually severe, is almost always relieved by amyl nitrite, though blood pressure does not always fall In the attacks the rise of pulse rate and of blood pressure begins before the pain starts evidence that the attack originates in a disturbance of the cardiac accelerators and the vasomotor nerves, whereby increased strain is thrown upon a defective heart But the relation between extra energy expenditure and pain is inconstant, and suggests that the vasoconstriction while raising blood pressure involves not only the abdominal but also the coronary vessels

DIAGNOSIS

The presence of clear signs of serious heart disease, and the severity of the attacks at rest, makes the diagnosis of a grave form of angina pectoris certain The associated disturbance of pulse rate and blood pressure may be suspected from the patient's story of violent throbbing but it should be confirmed or otherwise by observations in the actual attacks This malady is not to be confused with so called "angina vasomotoria" (page 72)

COURSE AND PROGNOSIS

The subjects who display angina of effort are for the most part middle aged or elderly when the malady first displays itself The malady is associated with processes in the vessels that are degenerative and usually progressive The usual course therefore is towards greater limitation of exercise by pain To avoid pain, less and less exercise is taken as time goes by, eventually pain comes with such slight effort that the patient is driven to his chair, his couch or his bed and is never secure from attacks of pain which now tend to be of increased severity A few pass into an anginal state and die in it, though this state more frequently results from coronary thrombosis The average life though very variable, is about ten years from the beginning of the attacks In very many cases the progress of the disease is slow, and men who experience their first symptoms in the middle forties live on to reach the middle fifties or sixties The course of angina is not always steady in its progression Years may pass with little or no perceptible change of condition or with considerable improvement In a number of instances the patients acquire signs of cardiac failure with or without fibrillation of the auricles Breathlessness rather than pain now curbs exercise These patients as a rule never suffer afterwards from anginal pain nevertheless the onset of failure with congestion in them is a very serious

event and terminates life usually within a year The fact that anginal patients are the subjects of general arterial disease and are prone to its various accidents, renal and cerebral, should be remembered The end does not always come through failure of the heart

The prognostic significance of angina of effort in individual cases should be regarded first from the standpoint of the amount of exercise needed to induce pain, cases that have mild pain on walking briskly uphill, pain that ceases soon if they come to rest, are in general favourable cases, those in whom pain of longer duration is induced by walking slowly on the flat are in general much more serious When the patient becomes sedentary in habit and experiences pain at frequent intervals, the end is not far distant Patients who have taken no care of themselves, have worked excessively, or have recently been subject to acute infection, are apt to hear a worse prognosis given than they deserve Judgment should [illegible]

elicited in the history or by observation The severity of pain on effort is not often of much value, apart from indicating the carelessness with which the patient nurses his malady An element of great uncertainty is introduced by the occurrence of coronary thrombosis, this accident cannot be foretold, but, though it may happen at any time, most of these patients escape it Sudden death will come to many of these patients whether they acquire a thrombosis or not, many will die of cardiac failure or of intercurrent maladies [illegible]

warrant Many men, senior in their professions or in public affairs, continue their busy lives for several or many years, enjoying immunity from all but occasional pain, by restricting their bodily activities Those who display the angina of effort are for the most part middle aged and elderly men, and it has been said that the expectation of life in these does not differ very greatly whether they have angina or not This is true of all but the more serious forms In considering what shall be said to the patient, it may be reflected that it serves no useful purpose to emphasise his advanced years, or to dwell upon the many ways in which an elderly or old, but otherwise healthy, man may lose his life

The particular syndrome, in which angina occurs at rest and

is associated with vasomotor disturbance and usually with aortic disease, is a grave one Although these patients may live precariously for a few years, spending much of their time in bed, sudden death is very common among them, it is in fact the usual ending.

TREATMENT

Rest and exercise —A principle of treatment is that the patient lives within the limit of his pain Outdoor exercise is beneficial and should be encouraged, but not such as induces pain When pain comes the patient should at once stand and rest and continue so at least thrice as long as it takes for the pain to disappear, he may then proceed, but a little more slowly, so that pain does not recur The benefits of exercise and the manner of its regulation should be explained to the patient, he may be told that, although his heart is not strong enough to carry heavy strains, such exercise as can be taken without symptoms re educates the heart and increases its strength He then has his own guide, and goes where he likes and does what he will within this limit, and he is thus comforted and encouraged to take a less gloomy view of the future than he otherwise would Reassurance prevents much unnecessary invalidism in apprehensive people Anginal cases should go early to bed and get up late Strenuous acts (page 291) should be forbidden for all time The patient should adopt strictly healthy habits (page 289). At least six hours' sleep at night should be ensured The patient is to be guarded against undue excitement, mental or physical, and from coldness in and out of doors The food should be small in amount, easily digested, nutritious and taken dry (page 292, Diet II), a rest of an hour should follow the heavier meals The use of tobacco should be forbidden, and not much tea or coffee should be drunk It is important to treat flatulent dyspepsia, for this is often responsible for attacks of pain or for their easier provocation If attacks of pain are easily induced, as by very quiet walking exercise, a period of several weeks' rest on couch or bed benefits Usually the pain disappears in such patients from the time they go to bed or within a few days, simply as a result of rest It is customary to find after several weeks' rest, and when the patient is up and about again, that more exercise can now be taken painlessly than previously It is as though there were originally some cumulative process in the heart, as though the muscle were in a sense tired, and recuperated with rest If attacks of pain are occurring apart from effort, very close enquiry

should be made to ascertain the cause the trouble may arise out of a concealed source of irritation or worry sometimes the patient will not recognise the cause until he is helped in his search for it

Many of those patients who work or exercise during the day, be come unduly fatigued or a little exhausted in early evening Rest and a glass of wine often comforts and revives them but they should do less Routine administration of vasodilator or hypnotic substance is not advised

Amyl nitrite capsules (2 to 5 minims or 0 1 to 0 25 c c) (or *nitro glycerine*) should be carried always by those suffering from angina of effort though they should not often require to use them the proper remedy is to cease exercising at the first warning of pain the nitrite is rather for the emergency for an attack of pain that is provoked unexpectedly or is of disturbing severity Alcohol may be used in similar emergency but the objection to its regular use in attacks will be obvious Those who are frequently forced to use nitrites should not be walking Attacks in resting people may be treated fully by nitrite and when severe and prolonged a number of cap sules may be broken and inhaled in succession until the paroxysm of pain ends or it is clear that the nitrite fails to take effect Often a single inhalation that flushes the face well suffices to stop an attack Throbbing under the drug is sometimes uncomfortable headache often follows its use patients whom it relieves may refuse it on these grounds The action of the drug upon the vascular system is fleeting but its effects on pain outlast this action Its potency is probably due mainly to its powerful dilatation of the coronary vessels rather than to its effect in lowering blood pressure It fails to relieve in cases where the coronary circulation cannot be increased as when there is thrombosis in the vessel supplying the affected area

Nitroglycerine (*glyceryl trinitrate*) is as efficacious as is amyl nitrite and is used in similar circumstances and causes much less throb bing and headache It is certainly the more valuable remedy in the stage when a progressive angina of effort has brought a man to his chair or couch it is then used to ward off attacks of pain that would otherwise occur as well as to lessen their severity The dose is $\frac{1}{100}$ grain (0 0006 g) and a tablet when slowly chewed and swallowed begins to act within one or two minutes and its action lasts for very many minutes This dose may be taken at intervals of a half hour or even less for periods of several hours if necessary The transaction of urgent business or the visits of friends are sometimes rendered possible only by its use it should not be employed with the idea of

simultaneously allowing greater bodily activity

Aminophylline (3 to 10 grains or 0 2 to 0 6 g , see page 40) may be given three or four times daily in cases with frequent attacks at rest, the smaller dose may be given intravenously in severe attacks This substance dilates the coronary arteries and is more prolonged though less certain in its action than the nitrites There are patients whom it benefits particularly, and who can continue its use in definitely Theobromine and caffeine are less potent

Ammonium bromide (15 to 20 grains or 1 0 to 1 3 g) taken thrice daily, for periods of a week or ten days with intervening periods of rest, is used similarly in the resting case with frequent attacks This method of dulling the nervous system is especially valuable in restless and sleepless subjects

Phenobarbitone soluble (½ to 1 grain or 0 03 to 0 06 g) twice a day may be used for the same purpose In the anginal state when severe attacks recur rapidly, morphia is used and on occasion light anaes thesia is required

Surgical interference —Removal of sympathetic ganglia has been proved to abolish the pain of angina in a number of patients It is a dangerous operation and I do not recommend it Paravertebral injection of the upper thoracic nerves with alcohol is safer It is an operation to be undertaken only by those skilled in the pro cedure and is to be recommended only in intractable cases in which severe pain occurs daily despite other remedies Thyroidectomy has recently been shown to relieve pain in a proportion of these patients total resection is practised, a low metabolism being established under control of thyroid gland

Special Cases

This and the preceding chapter describe the chief types of case in which anginal pain is the main symptom of a grave cardiac malady If these types are fully understood, few cases of grave

patients in whom the

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ontaining more women

than men, and well known to all medical men, they present the picture of long drawn and constant ill health These people never feel really well, they are depressed, often irritable, and in a state of constant fatigue They present a great variety of other symptoms, many of which are trivial, and, when all efforts to arrive at a more precise diagnosis have failed, become classed as neurotic Some of

these patients are actually suffering from unrecognised and chronic local infections, in others no such lesions are ever discovered The condition of the nervous system is such that any pain is poorly tolerated, it may be that the threshold for pain is low in these people Be that as it may, some complain of spasmodic pain referred to the chest and the state of the heart comes under discussion It is not to be forgotten that pain of anginal origin is more likely to be

its own basis Grave angina in women under forty five years of age should be diagnosed infrequently, and very rarely in younger women or much younger men who present no clear signs of cardiac disease, such as manifest enlargement of the heart or aortic disease Evidence of an anxiety complex, of constant ill health between attacks of pain, will naturally tend to deflect attention from the heart An incorrect diagnosis is often saved by detailed consideration of the pain, which is more frequently referred to the precordium than to the sternum, or is constituted by continual soreness, or is stabbing in quality Often in these respiration is rapid in the attacks, and the patient very restless Usually too there is no relation, or but a very imperfect one, between the pain and effort

Tobacco and angina.—The occasional heavy smoker is affected by attacks in which there is a sense of constriction in throat or chest, they may be accompanied by pallor, nausea, giddiness and weakness Occurring in early adult life, and ending when tobacco is discarded, they are probably to be ascribed solely to smoking An oppression in the chest or slight sense of sore constriction is not uncommon among heavy smokers when they take active exercise In elderly men it is probable, even when tobacco is evidently provocative, that there is an underlying predisposition to angina It is suspected, but not proved, that heavy tobacco smoking can lead to degeneration of arteries including the coronary vessels, it has also been supposed, though upon insufficient evidence, that excessive smoking can cause spasm of the coronary arteries

Tachycardia and angina.—Occasional cases of paroxysmal tachycardia, in which the rate of beating is high and paroxysm prolonged complain of characteristic anginal pain in the attack This example is interesting in that the heart is overburdened, not by any rise of arterial pressure, but merely by an excessive rate of beating

Severe anaemia, hyperthyroidism.—Attacks of anginal pain occur

sometimes in any severe anaemia and disappear when, under liver extract or otherwise, the anaemia is cured Here there seems a definite relation between anginal pain and nutrition of the heart It is not clear in this instance, or in the last of tachycardia, whether there is or is not an underlying disease of coronary vessels and a consequent predisposition to attacks of angina A similar statement applies to cases of hyperthyroidism, in which anginal pains may continue until the thyroid has been treated adequately

Vasoconstriction provoking thoracic symptoms —When adrenaline is injected intravenously into healthy subjects, pallor appears in the skin of the body generally and the pulse rate and blood pressure are raised In conspicuous reactions there may be strong palpitation and throbbing in the neck, a sense of constriction in the chest or actual pain across the sternum, closely resembling the anginal phenomena A corresponding group of symptoms and signs develops spontaneously in rare cases of paroxysmal hypertension, and has been described in association with suprarenal tumour, the attacks are thought to be due to natural adrenaline release, intense headache, nausea and vomiting may be added

Nothnagel described under the unfortunate term "angina pectoris vasomotoria" patients with similar manifestations The malady is a very rare one, so rare that no cases of precisely the same kind have been described since 1867, and it is here discussed chiefly for historical reasons Nothnagel's cases were not heart cases, they were chiefly men, otherwise healthy, and in their thirties and forties, in whom for a limited period attacks were provoked by exposure to cold or by bouts of drunkenness The attacks, which lasted a quarter to one hour, started with blanching of the skin, the hands and limbs being involved conspicuously and becoming numb and cold The pulse was narrow and small, its rate unchanged or a little slow These signs preceded anxiety or actual fear, and severe palpitation In one case, the only elderly subject of the series, pain of frankly anginal type appeared A few cases of a similar kind have been described with anginal pain but raised pulse rate

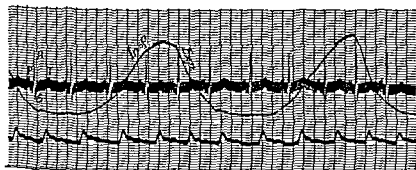
The real importance of the cases here grouped together is not that they confuse diagnosis, but that they suggest that vasoconstriction, by throwing a considerable burden on the undiseased, though not necessarily wholly efficient, heart, can determine secondary symptoms of considerable thoracic distress, or in the predisposed actual anginal pain

CHAPTER IX

MINOR PULSE IRREGULARITIES

Sinus Arrhythmia

The normal pulse may be definitely irregular in rhythm, this is frequent in childhood and adolescence, owing to what is called sinus arrhythmia *All the chambers of the heart are involved in the* irregularity, which though often inconspicuous may be pronounced It is nearly always related to respiration, the pulse quickening during inspiration and slowing during expiration (Fig 7) Occasion



ally the relation to respiration is indistinct, in such cases the nature of the irregularity may be tested by asking the patient to breathe *more* deeply, sinus irregularities *then* become more distinct and become obviously respiratory It is the rule for respiratory irregu larity to appear when young people breathe deeply These sinus irregularities result from inconstancy of vagal tone, and are almost

heart and a first, but not a second, sound is heard, the aortic valves have not lifted This is the extrasystolic intermission

In heart block, intermittence is produced by failure of the ventricle to respond to a rhythmic auricular beat (Fig 10) There is no premature impulse to detect, the heart is motionless and silent during the whole pause Thus the intermittence resulting from extrasystole is easily distinguished from that of heart block by attentive examination

Coupling and group beating—The pulse may intermit occasionally or at regular intervals If the regular intervals are close together, the pulse beats arrange themselves in simple groups Thus, if every third rhythmic beat is replaced by an intermittence, the pulse presents a coupling of its beats, if every fourth beat is

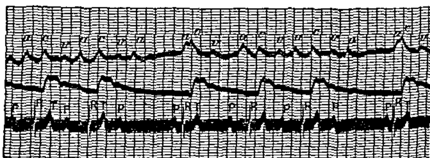


FIG 10—Simultaneous venous radial and electrocardiographic curves from a case of partial heart block. The *a c* and *P R* conduction intervals are double their normal values except after the longest diastole The ventricle fails to respond to an auricular contraction in two instances for in the venous curve an *a* wave and in the electrocardiogram a *P* summit stand alone Thus at regular intervals the pulse intermits

replaced, the pulse beats occur in groups of three *Coupling of the ventricular* beats will appear if an extrasystole replaces alternate beats of the ventricle, and the pulse will also display coupled action in the same circumstance if the premature beats are strong enough to yield pulse waves, but if the premature pulse beats fail, the pulse rate will be halved

It will be clear that very similar groupings of the pulse beats and halving of pulse rate can also occur when the ventricle fails to respond to the auricle at regular and frequent intervals

In distinguishing between these two mechanisms at the bedside we depend on being able to recognise the extrasystole, when it occurs, by remarking its prematurity and, when it is weak enough, by noting its failure to produce a second sound In cases presenting

difficulty the heart rate should be raised by exercising the subject, when the irregularity will generally disappear, a stethoscope is now placed on the impulse and the form of the first returning irregularity is noted In extrasystole it begins with a premature beat, in block it begins with an unusually long pause

Palpitation from extrasystoles —Symptoms may accompany intermittence or group beating when these result from extrasystoles, but not when they result from heart block In a large number of the subjects the extrasystole passes unnoticed, on the other hand it is a common cause of what patients term "palpitation" The symptoms are more prominent in the young, in women, in people of nervous temperament, or in general ill health or fatigue With the extrasystole there is a feeling of uneasiness, as of a void, in the chest, while the succeeding beat is accompanied by a shock to the chest wall, and often by a feeling of gripping in the throat By calling attention to the heart they awaken anxiety When numerous they may cause much discomfort, occasionally they bring actual distress, the skin becoming cold with sweat and the subject faint

Palpitation arising from occasional extrasystoles can generally be identified in a patient's account of himself, if he can clearly describe his sensations When extrasystoles are more numerous, the sensations are difficult to distinguish from those experienced in paroxysmal fibrillation (page 93)

Associations of extrasystoles —Extrasystoles are a very common phenomenon, occurring at all ages, but especially in the later periods of life, it is probable that they occur in most people who live into middle life or beyond it A few extrasystoles may occur each day, or from time to time, or they may persist for hours, days, or years Numerous or persistent extrasystoles are frequent in cases of chronic rheumatic heart disease, hypertension and chronic gastrointestinal disorders They may occur temporarily during the exanthemata or as associations of local infections They are often provoked by excessive smoking, sometimes by large doses of digitalis or its allies In those subject to them, extrasystoles are particularly prone to occur during rest after exercise, after heavy meals and at night shortly after going to bed In other subjects they occur only in the upright posture

Prognostic significance of extrasystoles —That extrasystoles are found in cases of serious heart disease and that the mortality among heart patients displaying them is high, are facts which provide no evidence that extrasystoles have grave significance The belief that

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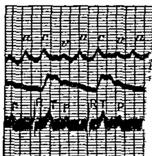


FIG 10—Simultaneous veno
heart block. The a-c and P R
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SYMPTOMS

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SIGNS ASSOCIATED

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tolic murmur are expected manifestations of a
n any circumstances

progress until extreme In the latter case the cardiac impulse continues its outward movement until it reaches the mid axillary line. In long-lasting paroxysms exhaustion is prominent In the last stages of steadily progressing attacks a cough accompanied by frothy and sometimes blood stained sputum develops with signs of engorgement and oedema of the lungs These steadily progressing attacks occasionally terminate in general dropsy, delirium, and death Almost all attacks end in a resumption of normal beating, in a very few sudden death occurs When the attack stops, the patient experiences immediate relief, breathing becomes free and the remaining symptoms soon disappear, the signs of engorgement disappear with remarkable quickness The heart resumes its original size within a few beats, the liver recedes more slowly in the space of hours Flatulence and weakness are often present for several days after an attack, and a sense of exhaustion and soreness of the chest may persist

In rare cases, and these usually known to be predisposed, a paroxysm may start an attack of characteristic anginal pain, slight, or severe and radiating

Auricular Flutter

This remarkable condition, in which the rate of auricular beating is usually as high as 260 to 320 per minute, is closely akin to auricular fibrillation It is believed to be caused by a wave of contraction circulating regularly and continuously through the auricle in this rapid rhythm (circus movement), it is due to a fault in the heart itself, the rate is so high that the ventricles rarely respond at more than half the rate (Fig 13) The usual ventricular rate is 130 to 160, that is to say, half the auricular rate There are rare cases in which auricular flutter occurs in short paroxysms, and it may then be confused with simple paroxysmal tachycardia *Usually, if untreated,* it continues unchanged for months or years The clinical associations and the symptoms of this persistent condition are similar to those of lasting auricular fibrillation (see pages 92 and 93) A history of fainting attacks is not uncommon in flutter

Diagnosis and Differentiation of Tachycardias

Cases of conspicuous tachycardia present themselves to medical men in one of several ways

A patient comes complaining of attacks of palpitation, and the

action of the heart is at the time normal The distinction between simple tachycardia and paroxysmal tachycardia, which is necessary in such a case, can often be formed on the basis of history alone The most important point in evidence of paroxysms is a perfectly clear history of abrupt onset and offset in the attack, with continuous rapid and regular beating during it, such histories can be obtained from observant patients On the other hand, a statement of gradual ending must be received cautiously, since it is sometimes given by patients whose long paroxysms end in a series of quite short ones or in a declining number of extrasystolic interruptions Another helpful point is the circumstance in which the attack occurs, although paroxysms of tachycardia may be provoked by exercise

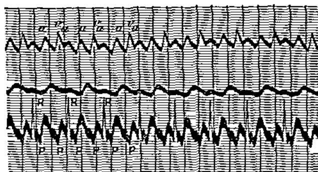


FIG 13.—Venous radial and electrocardiographic curves The record is from a case of auricular flutter in which the auricle was beating at 250 and the ventricle at 125 per minute The ventricular beats are recognized by the deflections R in the electrocardiogram one of which precedes each radial beat. These deflections R spring from a zigzag line the up-strokes of this line start at quite regular intervals and each complete phase corresponds to an auricular cycle alternate auricular deflections are increased in size because T deflections fall with them The venous curve consists mainly of very rapid a waves but alternate a waves are distorted by v waves being superimposed upon them. Time lines ⅕ sec.

or by emotion, usually they come unexpectedly and remain unexplained whereas in simple tachycardia definite acts usually bring them about or they recur regularly at given times such as meal times or on lying down at night The general picture will often point distinctly to simple tachycardia as when the complaint comes from a nervous woman at the time of the menopause But there will

ially be

When ever the heart rate in a patient at rest is found at 140 or over, the enquiry as to whether the tachycardia is of simple type or not

should be made The normal heart rate may be as high as 140 in an adult lying down and fully atropinised, occasionally and very *temporarily it rises to this* rate under emotional stress, more frequently to this or even higher rates in high fever and exophthalmic goitre But such rates are not long continued they fall as rest continues When, under resting conditions, the heart rate is fixed continuously above 140, essential abnormality of rhythm is usually *responsible When the rhythm of the rapidly beating heart* is normal, the rate rises when the erect, and falls when the reclining, posture is assumed, it responds by rising still higher when the subject exercises, and falls when he rests it rises higher in response to a whiff of amyl nitrite None of these changes influences the rate in paroxysmal *tachycardia, it remains at a fixed and high level* in a *great variety* of circumstances Very simple tests on patients, who are fit to submit to them, will thus distinguish the two states, but any test employed should be undertaken deliberately and repeatedly The beginning or the ending of an attack if witnessed should always be decisive, *for the change in the paroxysm is abrupt*

A patient is seen for the first time in an attack of rapid heart action, and the picture is that of acute failure with congestion It may be the first attack, in which case the help provided by accounts of previous incidents will be lacking The congestion being recognised, the condition is apt to be termed 'acute dilatation" of the heart, if the attack has begun during exercise, or while lifting a weight, it may be called "heart strain" Neither of these diagnoses is adequate, both are based on and lead to misunderstanding they do not appreciate the significance of the rapid heart rate and its consequences When in elderly subjects congestion and oedema of the lungs develop during the attack, dulness and crepitations at the bases may be mistaken for pneumonia Abdominal pain, anginal or hepatic, with signs of collapse and *running pulse in the attack*, have been confused on many occasions with acute abdominal symptoms Neither of these blunders will occur if the ventricular rate is accurately counted and the veins of the neck are carefully examined Forcible beating in the veins of the neck, with distension of the jugular bulb *on the right side, may closely* simulate innominate aneurysm on occasion

Flutter —When the ventricle is responding regularly and at half the rate of auricular beating the tests that differentiate flutter are the same as those that differentiate paroxysmal tachycardia from simple *tachycardia, for the rates* in flutter are also remarkably

constant in very varying circumstances The differentiation between paroxysmal tachycardia and flutter is not always easy in bedside tests In the former the rate is usually at or above, and in the latter at or below, 160 per minute A long continued regular ventricular action of 130 to 160 per minute in middle aged or elderly subjects should always suggest flutter If such tachycardia persists for two or more weeks and no change of rate occurs with rest or exercise, the diagnosis is practically certain Frequent repetition of precisely the same high rates in the pulse over periods of days, weeks, or months is an important sign On occasion and usually as the result of unusual emotion or exercise, the ventricular rate rises temporarily to the full auricular rate and syncope results It is often possible in flutter to slow the rate of the ventricle for a few seconds by pressure on the carotid sinus (see page 90), the original rapid rate being quickly resumed, this test is often valuable Digitalis given in adequate doses will always slow the ventricle and render its action irregular

When the original ratio of response is higher, for example, when the auricle is beating say at 300 and the ventricle at 75 (4 to 1 response), flutter is more difficult to recognise Occasionally the very rapid movements of the auricle are displayed in the neck and very rarely they are audible in the chest Sudden and precise doubling of heart rate (70 to 140 or 80 to 160) on slight exercise is significant

When the response of the ventricle to a fluttering auricle is rapid and irregular (Fig 14) the condition is usually confused clinically with fibrillation, unless exercise is found to make the pulse more rapid and at the same time regular The error is unimportant, for the treatment of the two conditions is much the same

The distinction between simple tachycardia, paroxysmal tachycardia, and auricular flutter can always be made with certainty electrocardiographically, in cases of doubt this method should be used whenever available

Prognosis

Simple tachycardia —The prognosis of simple tachycardia obviously depends upon its cause, and requires no further comment here

Paroxysmal tachycardia —In considering the immediate prognosis of the severe attack several aspects require emphasis The patient's symptoms are much influenced by the reaction of the nervous system, nervous subjects, especially women, awaken undue

anxiety The burden imposed on the heart depends upon the rate of its beating and upon the duration of the paroxysm, the time that a paroxysm will last can often be judged from the length of previous seizures The manner in which the increased burden is borne depends upon the cardiac reserve As a prognostic sign the strength of the pulse has little value Anxiety is apt to increase when, after several days, congestion is progressively increasing, respiration is becoming embarrassed, and dropsy is appearing But even when circulatory failure has become advanced, the paroxysm almost always ends favourably and the patient passes in a few minutes from a state of distress and of apparent danger to one of comfort and safety Confidence in recovery is almost always justified by the event, namely, an abrupt cessation of the attack Attacks lasting ten days are very rare, thus as each day passes the end of the attack becomes more inevitable A fatal termination is a rare event, except in those

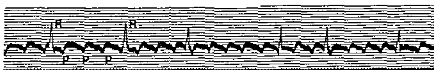


FIG 14 —Electrocardiogram of auricular flutter The auricular rate is 310 per minute the ventricle responds irregularly Time in $\frac{1}{5}$ sec

already gravely ill before the attack begins, from cardiac failure, pneumonia, or other cause

The future outlook of patients who suffer from paroxysmal tachycardia is naturally very varied It is based firstly upon an estimate of the soundness of the heart, and secondly upon the severity and frequency of the attacks The first factor is formed from the condition of the heart between the attacks The basis of prognosis is the same as in a similar case where there are no attacks, it being remembered that the attacks themselves indicate a defect in the muscle, and that very severe attacks temporarily threaten the patient's life The reaction of the heart to a paroxysm of tachycardia often itself provides valuable evidence of the heart's reserve When the heart is healthy a simple increase of rate (140 or more) causes its volume to decrease, whereas a defective heart reacts by dilating In the first case the circulation is carried on effectively and in the second it becomes impaired

There is no actual evidence that paroxysms of tachycardia leave damage behind them, though it would seem probable on general

grounds that frequent and severe attacks in subjects in whom there is little reserve must exercise an injurious effect The possibility of unexpected death in a paroxysm, though remote, necessitates some, but not undue, caution in the general prognosis

In general the most favourable, and incidentally the commonest, cases are those in which the heart is sound between the attacks and the paroxysms are infrequent, relatively short, and not severe Such paroxysms rarely shorten life Young subjects, and especially patients who are passing temporarily through a period of ill health, frequently lose their paroxysms altogether in later years The least favourable cases are those in which there is evidence of cardiac damage between the attacks and in which the attacks themselves are frequent and severe

Auricular flutter —Instances are known in which the auricle has continued to beat without cessation at 280 per minute for a period of fifteen or more years, the ventricle following suit at half that rate, and the patient meanwhile showing little change Prognosis follows much the same lines as it does in simple paroxysms and in fibrillation The reaction of the heart to flutter depends upon its reserve, and upon the nature of the burden that the flutter imposes It follows the lines laid down for fibrillation (page 96) more closely because both flutter and fibrillation are essentially persistent conditions, and because they arise in the same class of patient and present very similar ventricular rates that are in general below those of paroxysmal tachycardia In making a prognosis in flutter it is important to allow for the results of treatment, to which flutter when properly handled is very amenable

Treatment

Simple tachycardia —The treatment of simple tachycardia obviously depends upon its cause The rapidly beating organ is but a gauge telling of disturbed innervation, or of altered physical or chemical environment The discovery of an enlarged thyroid, signs of intoxication, a focus of infection, or instability of the nervous system forms the guide to appropriate treatment Bromides are most useful in treating attacks of simple palpitation, especially those associated with the menopause

Paroxysmal tachycardia —The treatment of cases of paroxysmal tachycardia resolves itself into remedies applied during the crises and general care of the patient during the course of the malady.

CHAPTER XI

AURICULAR FIBRILLATION (IRREGULAR TACHYCARDIA)

Introductory

It is believed that auricular fibrillation, like flutter, is caused by a wave of contraction circulating continuously through the auricle (circus movement), but differing from that of flutter by circulating over a variable path and completing 400 to 600 cycles per sec Like flutter it may be regarded as an intrinsic cardiac fault

In auricular fibrillation the normal quick and strong systolic contraction of the auricular walls is lost These remain in a position of diastole, and only fine tremulous movements, which have insufficient power to expel blood from the cavities, occur in them The auricular activity, though mechanically ineffective, stirs the ventricles to rapid and wholly irregular action, the rate of response, when the conducting tissues are quite healthy, being as high as 180 or even 200 a minute The usual rates of response are between 100 and 140 a minute In special circumstances the rate may be lower

Auricular fibrillation is one of the commonest forms of disorderly heart action in man Essentially it is a persistent condition, once established it persists for the rest of life unless interrupted by treatment A few cases are paroxysmal in type

Clinical Associations

In man auricular fibrillation has been noted from childhood to old age but it is extremely rare *before the age of twelve years* It is a frequent sequel to rheumatic fever or chorea Fibrillation accompanies mitral stenosis frequently, in aortic disease it is rare The rheumatic group of cases *is the largest*, and has its heaviest age incidence in the twenties and thirties A second group of cases of fibrillation display degenerative changes of heart and arteries, the

incidence being chiefly in the fifties and sixties At least 70 per cent of all cases of failure with congestion display fibrillation It is a common complication of hyperthyroidism (page 263)

PAROXYSMAL FIBRILLATION

Although fibrillation of the auricles may be spoken of as essentially a chronic malady, it occurs in some patients in attacks lasting a few hours or a few days Only in the very rarest circumstance is a paroxysm continued for longer than ten days The cases are usually rheumatic heart disease or hyperthyroidism, in others no abnormalities are to be found between the attacks Isolated attacks of auricular fibrillation sometimes supervene during the course of infection, especially acute infection, such as severe tonsillitis, acute cholecystitis, appendicitis pneumonia and infective endocarditis

SYMPTOMATOLOGY

[illegible]

symptoms at the onset of auricular fibrillation are similar to those already described for paroxysmal tachycardia and are similarly modified There may be no symptoms, or these may be severe Intelligent and observant patients will sometimes describe the beating of the heart as irregular As a rule, in persistent fibrillation, palpitation is not felt continuously but only when the ventricular rate rises high with effort or under emotion

CARDIAC REACTIONS

When the auricles fibrillate the heart works at a disadvantage In the first place the auricles are virtually paralysed, this, however, does not seem greatly to influence the general circulation In the second place, the ventricle beats irregularly and more rapidly Owing to the rapid action, the muscle enjoys less rest and it expends more energy (see page 30), the irregularity exaggerates this trouble because many of the beats are abortive, in that they fail *to raise the semilunar valves A relatively healthy* ventricle can tolerate the increased burden, a damaged ventricle does not, it dilates It should be clear from what has been said, here and previously, that the changes following the onset of auricular fibrillation,

whether this is the beginning of the chronic condition or whether it is to be a paroxysm, are in every respect almost identical with those described under paroxysmal tachycardia (page 83), and it is unnecessary to repeat them There are the same variations in the intensity of the symptoms, according to the rate at which the ventricle beats and according to the amount of the heart's reserve For a given rate of ventricular beating the disturbance is more intense in fibrillation than in paroxysmal tachycardia owing to the irregularity of the beat in the former condition The rate of beating, however, is usually less in fibrillation, but to offset this there is usually a smaller capacity of the heart for work Auricular fibrillation, when it comes, sometimes produces no increase of breathlessness on exercise, or only a slight limitation of respiratory reserve, but these cases are rare It is usual for the patient to develop persistent breathlessness, or a persistent increase of breathlessness, or shortly to be plunged into a state of failure with congestion, which will continue, if the state is unmodified by treatment, until death supervenes In cases that already show congestion before fibrillation comes, it will often immediately endanger life In elderly subjects the rate of ventricular beating is not usually very high, and so the change of mechanism more often passes unnoticed or without much disturbance

Cases of auricular fibrillation are relatively exempt, though not entirely so, from anginal pain

Recognition of Auricular Fibrillation

Auricular fibrillation is responsible for two series of signs, the one *is due to virtual paralysis of the auricle* the other to rapid and irregular action of the ventricle The auricular signs, comprising loss of the auricular venous wave and corresponding electrocardiographic deflection and the appearance of small waves derived from the continuously fibrillating muscle of the auricle, are well displayed in graphic records (Fig 15), by means of which auricular fibrillation is at once identifiable The only clinical signs that are produced directly from the auricle concern the effect of fibrillation upon the *heart sounds in mitral stenosis*, under which valve disease they are described (page 145) The diagnosis of auricular fibrillation without the aid of graphic records depends almost always upon study of the ventricular action Characteristically the pulse is turbulent, or grossly irregular, it consists of a medley of beats, varying in *force*

and most unevenly spaced It should be noted especially that phases of *irregularity are never accurately repeated*, and that when small groups of specially rapid beats occur these do not necessarily end in an unusually long pause Numbers of the beats fail to reach the wrist, but this is the habit of weak beats in general, as for example extrasystoles It is the rule for rapid beats to be weak, and, as rapid beats are numerous in fibrillation, many naturally fail to yield arterial pulsation The loss of beats in transit to the artery is unimportant to diagnosis, but makes it necessary that counts of ventricular rate should be taken from the heart itself

One of the most reliable clinical signs is based on the association of irregular and rapid action of the ventricle If the heart is beating

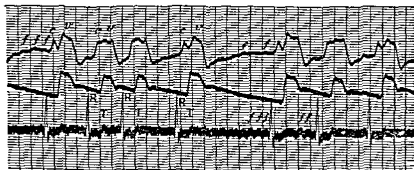


FIG 15 —Venous radial and electrocardiographic curves taken simultaneously from a case of [illegible]

irregularly and the ventricular rate is 120 or over, fibrillation is usually present the higher the rate in these circumstances the more definite becomes the diagnosis Sinus arrhythmia disappears when the rate surpasses 100, extrasystoles almost always when it surpasses 120, and the irregularity of auricular flutter almost always [illegible]

that are at all strenuous are undesirable, but in auricular fibrillation the rate lifts far more easily than it does when normal rhythm prevails Amyl nitrite may also be used on occasion to raise the rate in tests

The association of irregular heart action with venous congestion is in itself highly suggestive, but has not the same value as the signs described Auricular fibrillation is the only disorder that gives persistent irregularity of the heart's action other irregularities disappear occasionally, so that from hour to hour or day to day quite regular action is noted

In cases where the rate of ventricular response is low (below 80 or 90 per minute) the diagnosis is more difficult These are usually patients under the influence of drugs like digitalis, in such the presence of fibrillation will become more obvious as the effect of the drug lessens There are, however, cases in which the rate tends to be persistently low Very close attention to the arrangement of the pulse beats is then required, and the relation of the rapid beats to deep inspiration should always be investigated to distinguish fibrillation from sinus arrhythmia It is consoling to realise that although diagnosis becomes increasingly difficult as the ventricular rate is lower, so it becomes of decreasing importance, since it is the rapid action in fibrillation that calls mainly for treatment Actually, auricular fibrillation is diagnosable by the clinical tests here given in over 90 per cent of patients coming under medical care for the first time

PROGNOSIS

The prognosis of cases of paroxysmal auricular fibrillation is to be regarded from the same points of view as those of paroxysmal [illegible] the [illegible] nd [illegible] la [illegible] it should be regarded, not as a paroxysm, but as the chronic condition Untreated this lasts as long as life, the normal rhythm can be restored by quinidine in a number of patients but in only a few of these does it persist Normal rhythm is more likely to persist when the fibrillation was originally associated with hyperthyroidism and this has been treated (page 265)

The presence of persistent auricular fibrillation influences the general outlook in one of several ways No patient who displays it

can safely be regarded as possessing a sound muscle It may constitute the only sign that the muscle of the heart is affected and should always be so interpreted Auricular fibrillation loads the defective muscle with an extra burden, in considering this extra burden rate of ventricular response is of chief importance, high rate is not long supported by damaged ventricular muscle, but in this connection response to treatment is to be taken into account It is not the rate naturally prevailing, but the rate prevailing under such treatment as the patient can obtain, that matters A considerable reduction of the burden can be anticipated if adequate doses of digitalis are maintained, for this drug influences the ventricular rate in a remarkable fashion Nevertheless the onset of fibrillation in any patient is a serious matter, it is a particularly grave event in those already suffering from cardiac symptoms, at the least it means continuous medication, with the heart rate often ranging much above its previous average In most cases it heralds or actually brings venous congestion, few survive the event for more than five or ten years There are, however, rare but well authenticated instances of patients surviving for longer periods

Treatment

Paroxysmal auricular fibrillation is treated upon the lines laid down for paroxysmal tachycardia (page 89) Digitalis or its allies tend to [illegible]

[illegible]

as permanent and treated accordingly In the paroxysm a course of quinidine should always be given (see page 101), it is often successful Doses of [illegible] continued tend t[illegible] treatment the [illegible] mind in all paroxysmal cases

Chronic auricular fibrillation may be said to be the type of cardiac malady that when well managed benefits more from active interference than any other It is treated in one of two chief ways, by digitalis or an allied drug, or by quinidine

Digitalis Therapy

It is to their striking effects in cases of auricular fibrillation that drugs of the digitalis group chiefly owe their well founded reputation In fibrillation it is usual for the ventricular rate to be

increased digitalis can be made to control this rate as it can also in auricular flutter by decreasing the power of the auriculo ventricular bundle to conduct impulses from auricle to ventricle By slowing the ventricle it brings the heart its much needed rest from an unnecessary expenditure of energy There are few cases in which the desired slowing cannot be obtained and control of rate maintained by continued administration The higher the original rate of beating the more certain is the response to treatment The drug may be given beneficially whenever the heart rate is found higher than 80 or 90 per minute while the patient is at rest In treating cases of auricular fibrillation the guide is ventricular rate as this is counted at the cardiac impulse and not at the wrist

Patients treated for the first time should go to bed and remain there until the reaction to the drug has been investigated fully or for so long as other circumstances dictate As a routine tincture of digitalis should be used and if heavy initial doses are to be employed the tincture should be one that has been standardised biologically or by clinical experience in either case the stock of tincture drawn upon should be a large one and it should be known when the stock is replenished A usual and safe dosage for an adult is 60 minims (3 5 c c) of the *tincture*[1] *daily by mouth this may be given* as a single dose or divided into two or three doses It is continued until a reaction in the form of the desired slowing of the heart rate or until nausea vomiting or diarrhoea appear in response to the drug

It may be said that in the average a patient will use up or excrete 20 to 30 minims (1 2 to 1 8 c c) of tincture daily and that a reaction is not usually obtained until 5 drachms (18 c c) have been accumulated in the body so that the reaction is not usually seen before seven days Many patients require more and a few require less All must be watched closely As a rule ventricular slowing is the first clinical sign of the drug s action The object is to obtain a reaction including *a fall of rate to 70 or 60 per minute* If the remedy is maintained too long the rate may fall to 50 or even 40 and coupling of the beats may occur Neither digitalis coupling (Fig 16) nor profound slowing should be allowed to continue both are evidence of overdosage and call for immediate withdrawal of the digitalis Many patients at this stage seem to be in a most satisfactory state of convalescence they are actually in danger The patient should be kept at complete rest with strict avoidance of emotional excitement

[1] The approximately equivalent dose of powdered leaf is 6 grains (0 4 g) and of digoxin 0·75 milligram

until the heart quickens again I have seen more than one sudden and avoidable death result from neglect of the signs named In connection with slowing it is here to be observed that an allowance is to be made for the effects of fever upon the heart rate, the rate of the heart in this should not be forced lower than 80 or 90 per minute Coupling at high rates should rarely interfere with treatment, in such digitalis and quinidine may sometimes be combined usefully

In urgent cases, a more rapid reaction can be obtained by giving an initial massive dose of 2 drachms (7 c c) (to an adult of 100 to 140 lbs in weight), under which the heart rate will usually begin to fall within a few hours If there is little slowing, three fourths of this dose may be repeated next day and smaller doses given subsequently This method offers the prospect of rapid improvement, but is not to be used except upon patients under close observation, and



FIG 18 —The radial pulse curves from a case of fibrillation of the auricle exhibiting coupled beats arising from overdosage with digitalis The intervals between the large and small beat of each pair are uniform, but the long intervals which follow each couple of beats are very irregular in length.

only on patients to whom it is known no digitalis has recently been given It particularly suits cases displaying very rapid action of the ventricle (150 to 200)

Another plan, used in cases of the same type and with similar reservations is to administer the crystalline glucoside digoxin Either a dose of 0 75 mg is given in 10 c c of saline (alternatively, 0 25 milligram ($\frac{1}{250}$ gr) strophanthin), slowly and intravenously, in which case great care must be used that none of the drug escapes outside the vein or a dose of 1 5 mg digoxin is given by mouth The first begins to slow the heart rate almost at once and appreciably within two hours the second takes obvious effect within four or six hours Either may be followed by regular doses of digitalis at the rate of 1 drachm (3 5 c c) of tincture a day, starting the day after the dose of digoxin

There are patients in whom adequate slowing cannot be obtained by digitalis before gastrointestinal symptoms are induced by it Vomiting due to congestion of the stomach must be distinguished, because it often disappears under continued digitalis treatment, which clears away congestion vomiting is rarely produced by digitalis before 5 drachms (18 c c) of tincture have been taken Digitalis may be continued for a day or two advantageously in cases in which

it is causing nausea and occasional vomiting despite these symptoms, *for often the desired low rate follows soon upon these symptoms,* but dosage cannot long be maintained after they appear, and thus they very occasionally prevent fully successful treatment The nausea and vomiting can be relied upon to disappear soon after digitalis is omitted, both symptoms are largely due to effects on *the central nervous system,* but not exclusively so, they can be avoided sometimes by diluting the tincture and by giving it by mouth after meals only, or rectally Rectal or intravenous routes are indicated where the vomiting of congestion interferes with oral administration

Strophanthus is a much less certain remedy than digitalis and, owing to the difficult absorption of its active principles, must be given in doses which contain relatively large quantities of these if the drug is to be effective It has the danger too of variable absorption, and is very apt to cause diarrhoea, headache, yellow vision, and sometimes more serious symptoms Considering these undesirable characteristics and that it scarcely ever succeeds where digitalis properly administered fails I recommend its disuse by mouth In view too of the reliability and potency of digoxin, strophanthin is becoming less used

The initial object of treatment is to reduce ventricular rate to about 70 or 60 per minute the second object is to maintain the rate between about 70 and 90 beats per minute in circumstances of rest and light exercise When the rate has fallen on the initial heavy dosage,

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maintaining dose in patients out of bed is 30 minims (1 / c c) ot tincture of digitalis in the twenty four hours sometimes it is 20 (1 2 c c) sometimes it is 40 (2 4 c c) doses of 15 minims (0 8 c c) or less are nearly always useless for adults It is usual for the patient to continue such doses for the rest of life and to be seen every few weeks Continuance of the drug is governed almost entirely by heart rate, enough is to be given to maintain rates that are little in excess of normal *cumulative effects are not occurring if the rate is not* excessively slow Intelligent patients quickly learn to regulate their own doses by their sensations, taking the minimal quantity that frees them from breathlessness or palpitation they can be allowed to do so provided that a maximal dosage is named which may not be exceeded Business men who travel find it convenient to carry

digoxin tabloids (0 25 milligram) They are very potent, one or two a day should suffice to maintain proper rates for those already under the influence of the drug Although it is usually possible to control the rate in cases of auricular fibrillation, when the patients are at rest or quietly exercising, it is rarely possible to control the rates adequately in conditions of freer exercise It is unsafe to allow patients to exercise freely while upon full dosage of digitalis, and the ordinary "maintaining" doses will not prevent unusually high lifts of heart rate during brisk walking Thus, although patients with auricular fibrillation often return to work, moderately heavy manual work, strenuous games, and sports will always remain inadvisable For similar reasons women sufferers should be warned against becoming pregnant (see page 275) The patient should adhere to the rules of health laid down on page 289

Preparations of belladonna increase the rate of the ventricles and are contraindicated in auricular fibrillation

QUINIDINE THERAPY

Although digitalis is the safest and most reliable remedy in the treatment of cases of auricular fibrillation in general, some cases may be treated differently The drug quinidine exerts a powerful action on the heart, fibrillation can be abolished and normal rhythm restored by means of it, but it is a drug which should not be administered often It is only justifiable to use it in cases that are carefully selected and not as a remedy for cases of fibrillation in general Its administration is definitely forbidden in patients who have experienced haemoptysis or who present venous engorgement or much cardiac enlargement A very few patients develop collapse or syncope on the usual doses, so it is usual to give a preliminary test dose of 3 grains (0 2 g) in the form of sulphate If no unpleasant symptoms follow a course consisting of 6 grains (0 4 g) thrice daily, and twice, or better thrice, at night, may be used for a week, and the dose may then be raised gradually to double if the effects that are desired are not obtained The drug should rarely be continued for more than three weeks

It is usual for the ventricular rate to rise during treatment, and sometimes, but not often, severe palpitation prevents further administration of quinidine The development of headache, dizziness, and gastrointestinal symptoms rarely interferes, treatment should cease at once if urticaria develops, otherwise exfoliative dermatitis may follow Embolism following successful treatment has been a

not infrequent accident; it is due to clots becoming detached from the auricle when the walls of this chamber begin once more to beat vigorously. The risk of it is remote if the cases for treatment are properly selected.

I have found that cases most favourable for this treatment are those presenting few or no signs of heart disease other than fibrillation itself and especially those in which fibrillation has come recently. In all such it is my habit to give quinidine preference to digitalis, and this point of view is gaining ground. Some cases with mitral stenosis may be included, though they are less favourable In such selected cases normal rhythm can be restored in a very high percentage and is maintained in at least a third of these for years, with or without the continued use of quinidine. When quinidine is continued, a dose of 3 to 5 grains (0·2 to 0·3 g.) is given each morning and evening; this may be continued for years without adverse effects. Cases of fibrillation consecutive to hyperthyroidism are usually treated surgically (see page 265) and; if normal rhythm is not resumed spontaneously, quinidine will usually restore it, in such the drug need not be continued If cases of auricular fibrillation are less carefully selected, quinidine restores the normal rhythm only in about half those to which it is given, and the results are disappointing owing to the frequent early occurrence of fibrillation, and owing to the occasional occurrence of serious embolism.

CHAPTER XII

BRADYCARDIA, SYNCOPE, AND SUDDEN DEATH

Bradycardia

SIMPLE BRADYCARDIA

Simple bradycardia is a term used to indicate a natural action of the heart at a decreased rate, the whole heart participates in the slow action, and it is caused by decreased rhythmicity of the pacemaker (Fig 17) The heart in such is usually healthy In the human subject this slowing of the heart occurs in a variety of circumstances Although the rate of about 70 per minute has long been accepted as the average for the normal subject at rest, lower rates (65, 60, 55, or even 50) are not infrequent in perfectly healthy people The resting heart rate is often slower during adolescence (Fig 18) and is frequently much below the average in athletes The rate becomes slower with advancing years It is often decreased in convalescence from acute illness It may fall sharply after brief exercise and may be retarded by exposure to cold Gross cerebral lesions, such as tumour, apoplexy, and meningitis, are often associated with slow action of the heart, likewise myxoedema and, so it is stated, forms of insanity such as melancholia In jaundice the pulse is usually slow, often notably so In general the pulse tends to fall when blood pressure is raised Slow action of the heart is a usual accompaniment

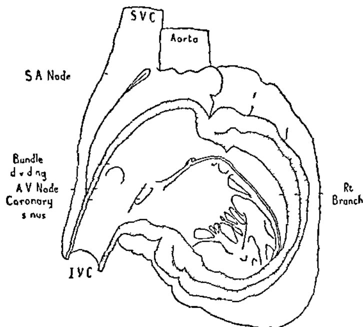
due to influences other than those of innervation

HEART BLOCK

Heart block is an abnormal mechanism in which the ventricle fails to respond properly to the beats of the auricle It is responsible for regular slow action of the ventricle when the latter responds

to each second beat of the auricle (2 : 1 heart block), or when it fails to respond at all and the ventricle assumes an independent slow rhythm (complete heart block) (Fig 19)

Heart block is caused by reduced power of the auriculo ventricular node or bundle, which normally convey impulses from auricle to ventricle, to conduct It may be produced experimentally (a) by direct interference with these structures, as by pressure or cooling (b) by inducing the vagus, and especially the left nerve, to



overact upon them and (c) by changing the character of the blood that supplies them, for example, by asphyxiation or by the injection of such substances as digitalis, large doses of adrenaline, or diphtheria toxin

In man heart block can happen at any age It may be congenital It often results from a lesion of the auriculo ventricular bundle In the young these lesions are mostly inflammatory, acute sub acute, or chronic, commonly the inflammation is rheumatic, some

times it is syphilitic; in later life degenerative processes with or without calcareous deposits are chiefly responsible Coronary arterial disease is the commonest single cause of high grade block Rarely tumours starting in, or invading, the bundle have been recorded The lesions that produce heart block are not often confined to the bundle

Minor grades of heart-block such as are responsible for intermittence of the ventricle (page 76) are commonly associated with infections, especially subacute rheumatism, diphtheria, influenza, and pneumonia The minor grades are often brought about also by vagal influences, as by pressure on the sensitive carotid sinus (page 90), vagal block is usually temporary and is relieved by atropine The same grades of block often occur during heavy administration of such drugs as digitalis, strophanthus, and squills, especially when these are administered to young subjects suffering from rheumatic heart disease

The only special symptoms associated with heart block are due to profound slowing of the ventricle or to sudden cessation of its beat, the resultant syncopal attacks are not infrequent in heart block of high grade They are described on page 111

DIFFERENTIATION OF BRADYCARDIAS

Differentiation of various forms of slow action of the ventricle may usually be accomplished by bedside signs The diagnosis can always be made absolute by means of graphic records In simple bradycardia the ventricular rate at rest is rarely less than 50 per minute In 2 1 heart block rates of 40 to 50 generally prevail In simple bradycardia the ventricular rate rises gradually with exercise, excitement, and such drugs as atropine and amyl nitrite In 2 1 heart block these influences frequently raise the ventricular rate abruptly to double and subsequently it falls abruptly to its original point 2 1 block is usually unstable, and abrupt changes of rate with or without intervening periods of irregular action are frequent Thus in differentiating simple bradycardia from 2 1 block such simple tests as exercise and the inhalation of amyl nitrite are very valuable

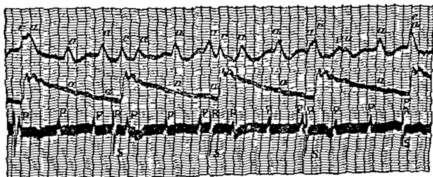
The diagnosis of complete heart block is suggested by regular ventricular action below 35 beats per minute, a constant rate of 30 per minute almost always indicates complete heart block, especially is this so if there is a history of syncope In complete block the rate of the ventricle is usually uninfluenced by exercise, atropine, or amyl nitrite The diagnosis can usually be made absolute by noticing the characteristic variations in the heart sounds which occur in

dependently of respiration. The breath being held, first and second sounds are heard with each ventricular systole; but these sounds vary greatly in intensity or show inconstant reduplication from cycle to cycle. Sometimes muffled auricular *sounds may be heard* over the right auricle, or at the impulse, or in the *epigastrium*. The



FIG 18.—Electrocardiogram showing the exceptionally slow heart rate of 36 per minute The *bradycardia* is a simple one of unknown origin, each cardiac cycle being represented by normal auricular and ventricular deflections Time in 1/5 sec

occurrence of numerous and regular waves in the jugular veins, some falling in the long intervals between ventricular systoles, is *also diagnostic*.



PROGNOSIS AND TREATMENT

Simple bradycardia is without direct prognostic or therapeutic significance.

Heart-block occurring under the influence of drugs should not influence prognosis; occurring otherwise, even in its milder grades

(dropped beats), it is to be regarded as a serious manifestation It is to be read as usually indicating a diffuse affection of the muscle, it should not be dismissed as a mere affair of the bundle, almost always the underlying process is widely spread throughout the more silent areas of the myocardium, and other evidences of a failing muscle are forthcoming In rheumatic fever, pneumonia, influenza, and other infectious diseases, its occurrence is significant of a severe infection and it may be the only outstanding sign of cardiac damage In most of such infections, however, the block is transient, there being recovery from much or all of the original damage Most patients who present persistent heart block die within a few years, whether the block is of milder or severer grade, and whether syncope occurs or not The majority never have syncopal fits but die of failure with congestion There are occasional cases in which heart block, even of high grade, continues for very many years, these are exceptional instances in which the driving muscle has suffered little and remains sound

Strictly speaking there is no treatment for heart block Treatment is directed to the cause or to the symptoms Where partial heart-[illegible]

manent The higher grades of heart block are usually persistent, the habits of these patients are governed according to the proved capacity of the heart to do work Many such patients are able to pursue usual duties, though they are unable or they are unwise to be very active Where there is any evidence that the lesion is progressive, rest and close attention are necessary Syphilitic subjects should be treated thoroughly with mercurials or arsenical preparations (page 229)

The prognostic significance of the syncopal attacks of heart block and the special treatment of these seizures are given more conveniently under syncope (page 114)

Syncope and Related Phenomena

An attack of faintness or actual fainting is a frequent reason bringing patients for advice It is a popular belief that such attacks are often manifestations of serious cardiac disease or precursors of apoplexy. Although there is some ground for these beliefs, the great

majority of the attacks referred to have far less significance It is a matter of much practical consequence that syncope and related phenomena should be understood thoroughly The chief forms of the attack referable to the cardiovascular system will be described, and subsequently contrasted with some phenomena from which they must be differentiated

By syncope we mean loss of consciousness caused by a deficient flow of blood from heart to brain The defect may be vascular the input of blood to the heart being diminished, or cardiac, the pump failing to do its work adequately When it is vascular there is a preliminary fall of general blood pressure A fall to 90 or 80 mm Hg may be maintained without effect on consciousness If the systolic pressure falls to such levels as 60 or 50 mm , the pulse becomes feeble or imperceptible Loss of consciousness occurs when systolic pressure falls to these levels or a little lower Syncope resulting from a decreased return of blood to the heart whether due to haemorrhage or to vasodilatation, is also associated with loss of pulse beat

POSTURAL FAINTNESS

If a hutch rabbit is held for a period by the ears its blood accumulates in the abdominal viscera, the heart may become empty and the rabbit shortly loses consciousness When the human subject passes from the supine to the erect posture there is ordinarily a small fall of blood pressure, which is quickly and in large part compensated In many people this phenomenon is exaggerated and leads to temporary giddiness or transient loss of consciousness on rising The complaint comes usually from middle aged or elderly subjects, and the symptom is particularly experienced on rising from a chair after a full meal in an overheated room On rising, the vision of the subject becomes dim, he is momentarily unsteady, puts out a hand to support himself, or braces his muscles and so recovers In some cases the effects are more profound and definitely alarm In rare instances the effect is not transient but continues, and the subject is unable to maintain both the erect posture and consciousness The phenomenon is due to an accumulation of blood mainly in the abdominal vessels, there is faulty distribution A closely related event occurs after stooping to lace the boots, or at the end of a strenuous lifting movement In both these acts blood tends to be driven out of the abdominal vessels by pressure or muscular action, and sudden resumption of the erect posture in the first, or relaxation in the second, instance leaves a

large potential reservoir for blood in the abdomen This then fills, meanwhile preventing a natural return of blood to the heart.

VASOVAGAL ATTACKS

These are the commonest form of fainting attack, they occur in both sexes, especially but not exclusively in the young A condition of poor health predisposes, thus they are more frequent after recent illness or in chronic infection, fatigue, a period of fasting, confinement to an overheated, overcrowded room are not unusual attendant circumstances The attacks are not confined to people presenting no signs of disease, they also occur in cases of chronic heart disease and in fact form the usual type of occasional fainting in aortic regurgitation and probably in aortic stenosis It is in sufficiently realised, as this example illustrates, that transient dis turbances yielding symptoms in relatively healthy folk also occur in cardiac patients and it is necessary to be on guard not to interpret such symptoms as belonging to the major disease The vasovagal attack [illegible]

confined to people who are unwell but also occur in robust health

Nervous agitation, emotional distress are commonly provocative, for example, an abrupt order or threat, an unexpected and important encounter, the receipt of unusual and stirring news, the witnessing of a distressing accident The provocation may be adequate without arousing feelings either of dread or disgust, there is usually an element of surprise, as when the subject sees his own blood flow into a syringe, or a sphygmograph lever begin to move on his own wrist The attack may be delayed until after the period of agitation is passed

The central feature of the attack is syncope, loss of consciousness may occur without warning and result in a heavy fall and injury, more usually there are warning symptoms, and then the subject sits or lies down deliberately, or slips or falls more slowly, the warning is a feeling of instability or uncertainty, a dimming of vision or giddiness Sense of rotation does not occur The attacks occur rarely, if ever, while the subject is lying down, but almost always while he stands or sits A peculiar sinking feeling in the abdomen, generally accompanied by nausea, sometimes by retching, and occasionally by a feeling that the bowels will be moved, are

often early symptoms Precordial distress or constriction do not occur, and real alarm is rare

From the beginning of the attack the blood pressure falls progressively and is usually accompanied by a steep fall of heart rate, the pulse often becoming imperceptible and the rate falling to 50 or 40 (exceptionally to 30) beats per minute Pallor, rapidly increasing in intensity, and ultimately extreme, accompanies these changes The pallor is of the type associated with simple drainage of blood from the skin, being most in evidence in the face and much less so in dependent parts such as the hands As cerebral anaemia increases the subject is seen to become unsteady, confused in speech, restless, and yawning There is decreasing response to questions and physical stimuli, loss of consciousness usually follows with flaccidity, some dilatation of the pupils, and lost conjunctival reflexes Slight clonic movements of face, upper limbs, and trunk may occur, respirations become slow, deep, and sighing Sweating, first of face then of body, is usual and often profuse

The attack is almost always brief lasting from two to ten minutes, rarely half an hour or more Recovery is slower than the onset leaving the patient limp and inert, and the skin pale and wet Weakness, tremulousness, and sometimes severe headache may last for hours

The slowing of the heart has been proved to be due to increased vagal tone, the slowing but not the loss of consciousness is relieved by atropine *The low blood pressure is independent of slowing, it is* due to vasodilatation and is responsible for loss of consciousness The double term vasovagal aptly describes two effects, proved to be dominant features of the attacks It is believed that the attacks are caused by reflex disturbance of a central nervous mechanism, which is normally set in unexaggerated motion by stimulation of either the carotid sinus (page 90) or the depressor nerve

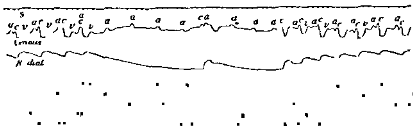
Carotid sinus and other reflexes causing syncope—In rare cases syncope results when light or moderate pressure is exerted over the carotid sinus There may be simple hyper irritability of the sinus, unexplained or following dosage with digitalis, or this region of the neck may be the seat of inflammation or tumour The symptoms are very similar to those just described, though sweating is less in evidence

Related are very rare attacks in which the vagal effects predominate These have been described as arising from irritation of diseased tonsils or oesophagus by food, of the respiratory passages by bronchoscope, or of an inflamed pleura during tapping The last has been known to end fatally.

CARDIAC SYNCOPE

Postural faintness and vasovagal attacks are common Instances of syncope, in which the heart fails to transfer incoming blood from the venous to the arterial side, are rare This is cardiac syncope properly so called, and results when the ventricles cease beating or beat very slowly, and when the ventricles beat very rapidly or fibril late It has no special association with any particular valve lesion

Ventricular arrest —Simple slowing of the heart fails to produce a material lowering of mean blood pressure, it raises systolic and lowers diastolic pressure, the heart accommodates itself to the in creased quantity of the blood returning to it in diastole throwing it out at each systole Great slowing of the ventricles alone gives rise to symptoms, in man unconsciousness results if the rate becomes



ca se was disease of the A V bundle

as slow as 20 to 10 beats per minute These rates are equivalent to cycles lasting about three to six seconds Cessation of the heart beat for a single period of five to ten seconds is sufficient to produce unconsciousness Arrest lasting from fifteen to twenty seconds causes obvious distension of the veins and deep breathing a little twitching of the face and upper limbs and cyanosis are added in pauses of longer duration

There are very rare cases in which the whole heart stops beating for a few seconds, these are usually vagal in origin In others, block between auricle and ventricle comes suddenly, normal response of the ventricle ending abruptly in a pause (Fig 20) Neither these varieties of ventricular arrest nor transient ventricular fibrillation (page 115), which is known occasionally to induce syncopal attacks, is of sufficient frequency to be of much practical importance

When arrest of the ventricular beating is responsible for repeated syncopal attacks, this arrest almost always occurs in cases of chronic heart block In patients who suffer from high grades of partial heart

block or in whom there is complete block attacks of giddiness, of momentary loss of consciousness, of longer unconsciousness with congestion and epilepsy, are common manifestations In these fits the ventricular action is extremely slow or ceases *for shorter or* longer periods the auricles continue to beat, as is shown by the waves that appear in the veins of the neck The severity of the attack is *governed by the degree of slowing or by the length of time over* which the ventricular beats lapse Death results when the ventricular arrest lasts a few minutes because the brain will not survive loss of blood supply of this duration In some instances of heart block the attacks may be absent or infrequent occurring at intervals of months or years in others they are more frequent coming weekly or daily or occurring in quick succession over periods of several hours or days and producing prolonged unconsciousness or a form of epileptic state When the ventricle stops beating the patient has no warning of the impending fit though on occasion a preliminary pulse slowing may induce momentary giddiness and this serves as a sign of approaching danger Incontinence of urine and tongue biting in the fits are rare Sweating is not a feature of the *attacks though* occasionally present in *them* and in *the stage of* recovery

Rapid action of the ventricles—Syncope may be caused by very rapid action of the ventricles such as occurs sometimes in auricular flutter (page 87) or in tachycardia arising in the ventricle It is usually necessary for the rate to rise to 250 or more At such rates *diastole is so short and the beats are so weak* that little or no blood is forced into the arteries and the blood pressure falls profoundly

Rare cases occur of brief loss of consciousness arising from a disorder which, if not ventricular fibrillation, is closely akin to it Fibrillation itself (page 115) is to be associated with sudden death rather than with syncope from which there is recovery

DIFFERENTIAL DIAGNOSIS OF FAINTING

It is often necessary to differentiate between fainting that arises from the heart or general vascular system—syncope as it is termed—and loss of consciousness that is primarily due to a cerebral condition such as epilepsy Syncope is always associated with loss of pulse beats, either because *the pulse becomes too weak to be perceptible*, or because the ventricle fails to beat Thus loss of consciousness, in which the pulse departs little from normal in rate or strength,

cannot be the result of diminished blood flow to the head and neck, it is a form of fainting that is cerebral in origin For similar reasons pallor is constant and conspicuous in syncope, it is not a feature of cerebral faints Clonic movements occur in syncope occasionally, but are not violent, thus tongue-biting and evacuation of the bladder, which distinguish many attacks of epilepsy rarely occur in syncope A sense of rotation never occurs in syncope, a fact that is very helpful in differentiating it from auditory vertigo

The peculiarity of vasovagal attacks, that they rarely occur in a patient who is lying down, is often crucial in distinguishing these both from epilepsy and from cardiac syncope Profuse sweating favours and is almost necessary to a diagnosis of vasovagal seizure, but does not exclude cardiac syncope, in which, however, sweating is rare The presence of heart block between attacks stamps these as due to ventricular arrest

Postural giddiness and vasovagal attacks in middle aged or elderly people are apt to be attributed to cerebral arterial disease, and regarded as attacks premonitory to apoplexy The cerebral arterial case usually presents hypertension, and the attacks may consist of dizziness without rotation, of faintness, or of actual loss of consciousness Postural giddiness can always be recognised by suitable enquiry The chief points differentiating the attacks of cerebral arterial disease from vasovagal attacks are absence of profound pallor or heavy sweating, and unaltered pulse in the former In attacks due to cerebral arterial disease, nausea may be present and headache is frequent, the association of loss of memory or inability to concentrate, paraesthesia, paresis or aphasia, renders the diagnosis of these seizures easy

PROGNOSIS AND TREATMENT

Postural giddiness is without any serious significance, it is occasional and not as a rule persistent It may be relieved or abolished by the use of a broad and firm abdominal support, which should be advised when attacks are frequent or sufficiently severe to threaten or induce loss of consciousness Otherwise treatment consists in attention to general health, to reduction of obesity, and to measures (exercise and massage) that increase the tone of the abdominal muscles

Vasovagal attacks are not serious They are occasional events experienced once, or a few times, and often in special circumstances that can be avoided In the attack the patient should be placed

CHAPTER XIII

CARDIAC ENLARGEMENT

The size of the heart is variable in healthy people, the weight of its muscle is related to that of the somatic muscle The taller and stronger the man, the bigger and heavier is the heart The heart is heavier in those who engage in heavy work or exercise than in those who live sedentary lives These are normal variations

Causes of Pathological Dilatation and Hypertrophy

Pathological enlargement of the heart may be due to dilatation of its cavities or to hypertrophy of its muscle

CAUSE OF DILATATION

Pathological dilatation is caused by inability of a well supplied heart properly to discharge its content Putting on one side exceptional cases of extreme obstruction to the outlet, inability to discharge is due to inefficient beating Inefficient ventricular beating may result from very excessive rate, as in flutter or fibrillation of the auricle It may be due to weakness of the muscle as a consequence of its poisoning, its inadequate nutrition, or its structural disease Examples of poisoning are to be found in the acutely dilated heart of such diseases as pneumonia, subacute rheumatism, diphtheria, and asphyxial conditions, of inadequate nutrition in coronary arterial disease, severe anaemias, and probably in instances of low mean blood pressure in the aorta, to which rate of flow through the coronary arteries is sensitive The pressure before which the heart dilates is that within it while it fills, it is a low venous and not an arterial pressure This applies to general dilatation, we here omit gradual distension of the cardiac wall where it is locally fibrosed—thus in the case of the ventricle a fibrosed area may gradually distend to form an aneurysm of the wall, which distends

under systolic ventricular pressure—and we here omit the events in rupture of the aortic valve discussed on page 162

CAUSE OF HYPERTROPHY

The recognised cause of hypertrophy is increase in the energy expenditure of the heart, sources of such sufficient to produce hypertrophy are persistent high blood pressure, defects of the valves or septa, and in certain instances pericardial adhesions

Increased load falls on different parts of the heart unequally. Thus mitral stenosis imposes its burden upon the left auricle and right ventricle, and these are the chambers that show corresponding hypertrophy most conspicuously Aortic regurgitation and aortic stenosis burden the left ventricle and cause most conspicuous hypertrophy in this chamber High systemic blood pressure is the most potent cause of left ventricular hypertrophy The distribution of these hypertrophies, however, is not so simple as it is often thought to be Thus, in half the cases of mitral stenosis the left ventricle is increased quite as much as is the right, and in half the cases of aortic disease the right ventricle is increased quite as much as, or more than, is the left In both diseases it is the rule for the two ventricles to participate in the enlargement, although one usually does so more than the other Simple adherence of the visceral and parietal pericardium produces little or no enlargement of the heart, the burden of the heart may be increased however, when the heart's action is impeded by adhesions between it and surrounding structures There are many cases of hypertrophy, and of great hypertrophy of the heart, in which during life and after death no source of increased work can be discovered The degree of enlargement found in many hearts in which there is a valve defect, such as aortic regurgitation or mitral stenosis, is sometimes out of proportion to the apparent increase of burden There is still much that remains to be explained, it is clear that there must be hidden sources of increased energy expenditure, or the idea that increased energy expenditure is the only cause of hypertrophy is wrong

SYMPTOMS

There are no known symptoms of hypertrophy of the heart Such symptoms as might be ascribed to dilatation have been described already under failure with congestion

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tion of the heart beat, though producing a diffuse impulse in the spaces, rarely moves ribs or cartilages appreciably Prominence of the precordial ribs and of the lower sternum is frequent when the heart has become much enlarged during the period of growth before the chest wall has hardened to its final shape

Epigastric pulsation occurs in one of three distinct forms It may be transmitted from the abdominal aorta and is then felt as a forward systolic thrust, occurring a little later than the cardiac impulse It may result from pulsation of the liver and is then visible but rarely palpable, and is associated with abundant signs of venous congestion It may be transmitted from the heart, in that case the movement is almost always a systolic retraction, and pulsation is felt against the upper margin of fingers pressed into the epigastrium This, the usual cardiac form of epigastric pulsation, is valueless in estimating the size of the heart, being produced very readily by overaction of the heart, and occurring in many perfectly normal people In cases of hypertrophy of the heart, a strong downward systolic thrust from the heart is sometimes felt by fingers that sink into the epigastrium

Percussion —This method of measuring the heart has much value if used reasonably It is, however, a highly subjective method and has an error that is often considerable It is crucial in measuring to know the error of the method, to have but an inaccurate measure may be regrettable, but to have it and not to know it is deplorable Very fantastic ideas have been, and still are, held of the accuracy that can be attained by percussion It is illuminating to practise percussion while blindfolded and repeatedly to percuss in a rib space chosen by some critical observer, who chalks the margin of dulness on the chest wall as it is estimated It does not require much of this experience nor long comparison with X ray records to know that great accuracy is impossible The left wall of the heart often moves in by as much as 1 centimetre in systole, it often moves more than this with respiration Yet we take no heed of the phase of the cardiac or respiratory cycles in which strokes are delivered A left margin of dulness coinciding with the maximal impulse is usual enough, and to be expected, since this impulse often forms the outermost point of the heart that is uncovered by lung The impression of a sharp margin of dulness in other rib spaces is mostly fictitious, this must be so in that there is a gradual recession from the chest wall as the intervening lung tissue gradually increases in depth The idea that percussion gives a projected image of the heart could

only apply to small hearts percussed on the front of the chest When the heart is enlarged percussion wanders farther into the left axilla and the stroke is now towards the side of the heart and not towards its front, it is not a margin of the heart that is being percussed Margins of the heart are purely imaginary, its walls merely project certain distances from the mid line in given planes It is incorrect to speak of the border of the heart All we can expect to do by percussion is roughly to map out the region of the chest wall that is *approached rather nearly by the heart and by the great vessels* An increase of the cardiac dulness to the left is an important diagnostic aid, but should never be allowed to outweigh the evidence of a well defined maximal impulse Thus the chief value of percussion to the left is in bringing confirmation when the impulse is ill defined, and in ascertaining the approximate extension of the heart towards the axilla, when no distinct impulse is available

In marking out the position of the heart by percussion, moderately firm percussion should be used The left border of dulness is ascertained by percussing the 5th, sometimes the 6th, intercostal space, starting well out in the axilla, and working forwards and inwards in the chosen space The outermost normal limit is 4½ inches (11 cm) from the mid line

In marking out the right border of dulness, the upper border of the liver to moderate or rather lighter strokes is *first ascertained in the* right nipple line Percussion is now conducted inwards immediately above this liver dulness Dulness corresponding to the right auricle is generally encountered 1½ to 2 inches (3 to 5 cm) from the mid line, in the 5th interspace The chief information to be obtained from the right border is that the heart is or is not displaced Increased dulness to the right occurs in cases of auricular enlargement, especially in mitral stenosis

It is usual to percuss the upper border The method has little value and it is open to the objection that the upper border is obliquely set It is of much more practical value to percuss as a routine the 2nd left space from left to right and to cross the sternum The 3rd left space [illegible] ld be in special cases [illegible] nd its percussion will [illegible] ac dulness upwards [illegible] Simultaneously it reveals the dulness of a dilated aorta or pulmonary artery, which under the ordinary routine of percussion is often overlooked.

Acute Dilatation

When the heart dilates in a paroxysm of tachycardia it chiefly bulges laterally, and more to the left than to the right Thus a main sign of acute dilatation of the heart is movement of the impulse and of the left border of dulness outwards, the movement is more important than the position the impulse has finally adopted Extreme dilatation of the heart carries the impulse well out into the left axilla (see pages 83 4) In acute dilatation the impulse may become diffuse, though diffusion is commoner in chronic enlargement Another frequent associated sign in the early stages of dilatation is the development of a blowing systolic murmur in the region of the heart's apex It is erroneous to believe that the rapid heart action, often associated with dilatation, is caused by dilatation (see pages 83 and 86) Most, if not all, instances of appreciable cardiac dilatation are accompanied by congestion of the veins It is not yet clear that there is a venous pressure corresponding to each grade of dilatation, though it is clear that there can be no increase of pressure in the right auricle that is not at once reflected as a corresponding rise of pressure in the systemic veins It is only in these veins that the measure of increased pressure can be taken clinically

Right and Left Hypertrophy

It is in general true that a heaving lower sternum means a hypertrophied right heart, and that a heaving impulse in the 5th or 6th

and a heaving impulse in 6th or 7th space in the left axilla may come occasionally from the right ventricle When there is much enlargement of the heart in a case of chronic disease, it is safest to assume that both ventricles are involved The attempt to distinguish clinically is a diagnostic affectation, it is rarely practicable and never very helpful Relative preponderance of the two ventricles in cases of known enlargement of the heart may be determined electrocardiographically (Figs 22 and 23), even so, the information obtained rarely influences either prognosis or treatment

Great Dilatation of Left Auricle

An aneurysmal dilatation of the left auricle (500 to 1000 c c) [illegible] in rare cases Usually these patients give a history of rheumatic [illegible]

nearly all show auricular fibrillation, more than half of them present mitral stenosis and most of the remainder some thickening of the valve and widening of the ring The auricular wall is often of the thinness of paper, and fibrous tissue replaces its muscle It is because the muscle has perished that the dilatation occurs The left auricle, in expanding, bulges to the right behind the right auricle, which normally lies in front of it, into the lower right pleural cavity The condition is important in that it may be mistaken for pleural or pericardial effusion and be tapped Dulness is extensive to the right

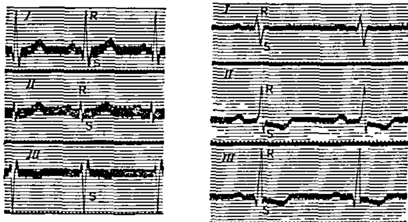


FIG 2° —Electrocardiograms from leads *I* *II* and *III* illustrating preponderance of the left ventricle *R* is tallest in lead *I* and shortest in lead *III* *S* is deepest in lead *III*

of the sternum, from the 4th rib or space, the border of dulness *extends as a convex line outwards and downwards* through or beyond the right nipple the whole of the lower axillary region or even the base may be dull Other signs helping to differentiate the *condition from effusion* are cardiac pulsation and clearly audible heart sounds over the anterior area of dulness X rays show the enlarged auricle and bowing of the oesophagus to the right and *backwards*

Distinction between Hypertrophy and Dilatation in Chronic Cases

In considering whether hypertrophy and dilatation can be separately assessed in chronic heart cases during life, we deal with

questions of measurement The only reliable way of measuring the amount of muscle in the heart is to weigh it after death, and this is our measure of the hypertrophy that was present in life

In measuring the size of the heart during life we are measuring the size of the organ with its blood content Our gauge is one relating to the heart's volume By measuring in several planes we might arrive laboriously at an estimate of volume, this would include the volume of the muscle and the volume of the contents Volumetric measures after death are fallacious, because the heart is very apt to distend in dying, and because much blood may run into it after it has ceased to beat

How is dilatation to be gauged? If a heart of normal volume swells acutely, then the measure of increased volume is a simple measure of dilatation The dilatation that happens during a severe paroxysm of tachycardia can be gauged approximately by repeated observation during the progress of the paroxysm But no estimate of the amount by which the heart has dilated can approach accuracy if it depends upon observation confined to the end of the attack, since the original size of the heart will be unknown Thus, in a case of chronic cardiac enlargement, our only hope of obtaining an accurate measure of the dilatation during life is to estimate the volume of the heart during life, and, a little later if opportunity offers after death, to subtract from this the volume of the muscle Hypertrophy can be measured after death, volume might be measured during life Dilatation could be measured by combining these two, but it has not been done It is to be observed that physical signs, purporting to indicate degrees of dilatation in chronic cases, may not be introduced into the argument, for any that we may possess are unreliable in that they remain unchecked against a proper measure We cannot assess hypertrophy or dilatation where both occur together, with any real accuracy, either during life or after death

"Compensation"

More than a hundred years ago the view was put forward that when a heart has gained, in virtue of its hypertrophy, as much as it has lost by reason of its dilatation, there results a sort of compensation or equilibrium, which maintains the cardiac function in its normal state The view, thus expressed, gradually became more dogmatic, governing the interpretation, and even influencing the recognition, of cardiac failure If it were held, in the case of aortic regurgitation, that hypertrophy is a process of "compensation",

enabling the heart to meet the added load, no exception could be taken to such a use of this word But the word stands for a fuller hypothesis It is that if dilatation appears, the circulation is imperilled, and that the danger continues until a corresponding degree of hypertrophy is added, then the balance is restored, and the heart again functions normally The later appearance of adverse symptoms is heralded as "broken compensation" and implies that the balance between dilatation and hypertrophy is again upset It is to be presumed that the balance is upset by a gain of dilatation rather than by a loss of hypertrophy The hypothesis is open to grave objections, for this reason, and also because it has influenced diagnosis prejudicially, it is necessary that it should be abandoned as a clinical tenet

Knowing, as we now do, that the normal heart in meeting extra work dilates, and thereby increases its capacity for work, we possess a clear warning that the hypothesis is not unassailable, for the essence of the hypothesis is that dilatation is deleterious Again if it is agreed that every breach of compensation is remedied by a corresponding hypertrophy, it is clear that each event of this kind must leave the heart larger Now this is definitely not the case it is not necessarily the case even after repeated attacks of cardiac failure with congestion The fact is that it is not dilatation that is injurious but that which is responsible for dilatation Dilatation of the heart is the result rather than the cause of failing efficiency When a patient suffering from fibrillation of the auricles loses his venous congestion under digitalis, he does not do so because hypertrophy develops and balances dilatation, neither does he do so because dilatation of the heart disappears Venous congestion disappears because with the lower rate of beating the heart acts more efficiently, and if the heart shrinks, as it may sometimes in the process, this is not the cause of a restored balance but a secondary effect of longer diastoles and more powerful systoles The use of the term "compensation" at the bedside conjures up or fosters the belief that, by signs derived from the chest wall, judg

pensation' and broken compensation, or its ugly [illegible] compensation", at the bedside and mean little more than to infer that venous engorgement is absent or present, but that is not what is to be understood A plain observation is garnished with an assumption

and served up in an unrecognisable form, this too prevalent habit is pernicious, for it destroys simple and precise habits of thought Terms involving unproved hypothesis should be eschewed, and in this instance reference should be to the simple presence or absence of venous congestion By this directness, cumbersome, needless, and in fact erroneous, assumption is avoided, and simplicity and exactitude of expression are secured The diagnosis of venous congestion is within the easy reach of all, it is a simple expression of what we see. The diagnosis of "broken compensation", in its proper sense, is within the reach of none

Degrees of Chronic Enlargement

It has been seen that an estimate of the relative degrees of muscular growth and stretching in chronic enlargement of the heart cannot be made Moreover, it is unnecessary, in the sense that the goal, at which the attempt is aimed, is reached more quickly and more certainly in other ways It is probable that in most instances of permanent enlargement, hypertrophy and dilatation coexist and are more or less proportioned to each other, for weight can be guessed from the volume of the heart without frequent and gross error during life The chief example of inaccuracy is the instance in which a heart definitely pronounced small during life weighs much more after death than was expected

In patients suffering from, or examined to exclude, chronic disease of the heart, we can undoubtedly estimate the size of the heart in a broad way in most instances by simple bedside signs Great accuracy in measurement, as we have seen, is unattainable, were it attainable it would serve no very practical purpose It is rarely a matter of much consequence to treatment whether we diagnose the heart of normal size or very slightly enlarged The idea that a fine gauge is needed is erroneous, a coarse gauge suffices If the right border is found in its proper place, and the outer margin of the impulse and left percussion border lie 4½ inches (11 cm) or more to the left of the mid line, or if these lie a lesser distance to the left and there is unmistakable thrust of bony or cartilaginous structure, *definite enlargement* (meaning slight but unmistakable enlargement) of the heart can be diagnosed If in similar circumstances the left border lies farther out, and the impulse farther out and perhaps down, and there is perhaps heaving of ribs or sternum, *moderate enlargement* can be diagnosed When the impulse and percussion border are far out in the axilla, and there is forcible movement of

much of the chest wall, *considerable enlargement* can be diagnosed The subdivision is an arbitrary one, and subject to minor modification, but it serves to place patients, from the standpoint of the heart's size, in a few simple categories which suffice for all practical needs It is far better to concentrate upon this relatively simple end result than to attempt fine distinctions of more than doubtful value

Prognosis

Dilatation —It follows from the manner in which pathological dilatation of the heart is produced that it is significant of inefficient beating as this is produced diversely, so the outlook varies, for the manner in which dilatation is brought about must be taken into consideration When dilatation is due to very rapid heart action, prognosis is really concerned chiefly with this rapid action and its cessation for when the heart begins to beat slowly again it will cease to beat inefficiently Dilatation in itself does not spell disaster unless it is extreme and extreme dilatation is usually prevented by an unstretched or normal pericardium The circulation can be carried on for long periods as paroxysmal tachycardia illustrates while the heart is much dilated, and recovery is prompt and complete at the resumption of normal beating Similar considerations apply when the heart is dilated during infectious illnesses, the dilatation is a measure of the severity of the poisoning, appreciable dilatation occurs only when intoxication is reaching a dangerous level It is natural to regard such a condition as grave, but attention should not concentrate on the heart other parts of the organism are simultaneously suffering The seriousness of the situation lies in the course the infection is taking rather than in the dilatation itself Dilatation as seen in infectious disease rarely reaches the grade that may occur temporarily in paroxysms of tachycardia, except in the terminal and hopeless stages of the infection These examples sufficiently illustrate prognostic considerations in acute dilatation of the heart

Enlargement of the heart in cases of chronic heart disease is an important guide to prognosis In considering this matter it is in [illegible]

valvular disease, similar exercise tolerance, etc , but differing only, though differing clearly, in the degree to which the heart is enlarged Tested on these lines it is evident that enlargement is significant

and it is significant according to the degree in which it is displayed In the chronic disease of adults it is remarkable how little the actual physical signs of enlargement change from year to year Enlargement is not a steadily progressing malady Enlargement often comes in the early stages of the disease and remains fixed in its degree Hypertrophy in response to a constant new burden is held to develop fully within a few weeks The reasons why enlargement affects prognosis are that enlarged hearts are far more prone than are small ones abruptly to display signs of failure, that they are more apt to suffer from thrombotic accidents coronary or endocardial, the latter leading to embolism, and that they tend more to acquire serious disorders of beating It has long been held, and I think rightly, that the muscle of a greatly enlarged heart is essentially unsound, but this statement is based, not upon histological or experimental evidence, but upon the experience that large hearts are not dependable

The place taken by enlargement in the prognosis of chronic heart cases in general is given in the final chapter

Treatment

Treatment in cases of acute dilatation should be governed by the origin of this dilatation If it arises from a paroxysm of tachycardia, then the paroxysm is to be treated, once this has ended and as usually happens, the dilatation has disappeared completely within a few heart beats, the fact that the heart has been dilated does not call for special treatment Patients who feel exhaustion will want to rest for a day, others will soon feel quite well and may be allowed to go about their ordinary affairs If the dilatation results from an infection then it is the latter to which the remedies must be applied and which by its course and severity will dictate the period of rest If it comes from asphyxia, then any obstruction of the respiratory passages that is present must be removed These are the only common ways in which acute dilatation of the heart arises and treatment corresponds There is no common acute condition to which we can suitably apply the term idiopathic dilatation[1] or that calls for special treatment and after care (for Heart Strain see Chapter XVII) but if acute dilatation is so severe as to bring with it great venous engorgement, then, if it cannot be attacked at its source, special remedies for congestion may come under considera

[1] Beri beri heart as it has been termed is a malady of the East Its increasing diagnosis in this country exemplifies the current itch to vaunt vitamin deficiencies

tain a full circulation, such blood as regurgitates must be thrown out again as well as the normal ventricular content Thus, an increased quantity of blood is ejected rapidly into relatively empty arteries, especially when regurgitation is free The top of the pulse, which represents systolic blood pressure, is high The pulse pressure (the difference between diastolic and systolic) is great

SYMPTOMS

There are few symptoms that belong properly to incompetence of the aortic valve At the most we may recognise those that arise from the unusual momentum of the issuing blood and from the rapid expansion of the arteries Consciousness of the heart beat (palpitation) is usual and may be severe Throb is often felt in the neck and head, and beating is heard in the ears There may be a general throb of the body with each heart beat The momentum of the blood discharged may suffice to shake the bed or couch on which the patient rests and he hears it The patient may be aware of a rhythmic noise within the chest after the aortic valves have ruptured It is doubtful if faintness while standing is commoner in these than in other heart cases

SIGNS

There are two signs that stand out as of chief value in diagnosis, and these are the characteristic steep rising pulse of free regurgitation and the diastolic murmur at the cardiac base With both these signs present the diagnosis is certain, with either one or other it is probable

Water hammer[1] *pulse* —In free aortic regurgitation the quantity of blood ejected at each beat is in excess of normal, and it is ejected by a powerful ventricle into an aorta in which pressure is unusually low, the pressure therefore rises with abnormal rapidity and to an unusual height Thus, the characteristic pulse is large, jerking up abruptly and representing a greatly increased range of pulse pressure The upstroke of the pulse is much steeper than that of the normal pulse (Fig 24) and steeper still if the wrist in which it is felt is held aloft If the whole wrist is grasped with the fingers lying over the front of it, the normal pulse is scarcely felt, but the aortic pulse slaps the examining fingers sharply and gives often a distinct and correct impression of an accompanying thrill

(Fig 24) All the pulses of the body jerk forcibly, as does the radial, and the movement is clearly to be seen over the line of the carotid and brachial arteries The carotid artery may move the lobe of the ear and the pulsation be continued actively into the superficial temporal vessel The abdominal aorta throbs violently As it travels, the pulse wave often rises more steeply and to a higher crest, and so it frequently happens that the abrupt jerking (or water hammer) quality is extreme in the dorsum of the foot, for the same reason the systolic pressure comes to be higher, often much higher,

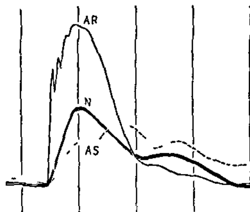


FIG 24—Tracings from three selected optical records from the radial pulse The highest curve is the pulse of a case of free aortic regurgitation (*AR*) and is characteristic it is of extreme abruptness in its upstroke which continues in a series of oscillations to the summit it is a perfect example of a water hammer pulse

The lowest curve (*AS*) is from a case of aortic stenosis and is anacrotic in form rising slowly over the shoulder to a very delayed summit.

The third curve (*N*) is a normal curve for comparison with the remainder The vertical lines represent time in ⅕ of a second

in the leg than in the arm Many other signs are recorded that belong to the same category, for example the "pistol shot" sound, heard when a stethoscope is pressed over an artery, and the audible pulse in the palm, there is no need to cite them all, for they express one and the same phenomenon and are in reality redundant signs

Because the summit of the pulse rides high, the fall of pressure to the dicrotic notch is more rapid than normal, a feature that has led to the use of the name "collapsing pulse" Actually, exaggeration of the rate of fall is far less than is exaggeration of the rate of rise, and cannot be detected with the finger, the term "collapsing" detracts attention from the essential quality of the aortic pulse and should be abandoned.

The pulse in aortic regurgitation deserves close study, not only because it is so important for diagnosis but because it provides the only real guide to the degree of reflux From the full arterial signs described there is a complete transition to the arterial signs that appear in healthy people, in very slight aortic reflux the pulse pressure is normal in extent and the upstroke normal in rapidity

Diastolic murmur —The characteristic murmur of aortic regurgitation begins abruptly in early diastole and fades away It is usually a long, smooth, rather loud, blowing and high pitched murmur, sometimes it is much shorter and far less audible and requires very close attention to detect it It is heard best over or near the sternum at the level of the 2nd rib or space to right or left of the sternum, or in the 3rd or 4th spaces to the left of the sternum The stereotyped phrase that the murmur is maximal at the 2nd right cartilage is inaccurate *It is also often heard along the right border of the sternum*, or may be traced over the precordium as low as the cardiac impulse *In the region of the 2nd rib it is almost always* preceded by a systolic murmur whether stenosis is present or not It is instructive to note, as a comment upon the timing of murmurs, that there is no murmur that gives rise to more frequent differences of opinion, or to more changes of opinion by one and the same observer than this diastolic murmur when isolated, and especially when the heart is beating a little rapidly Happily, the systolic murmur rarely does fail If the stethoscope is placed over the 2nd right cartilage, and to and fro blowing murmurs are heard, it is sufficient there is then no need to attempt the timing of these murmurs—the first is necessarily systolic and the second necessarily diastolic The diastolic murmur is often heard more loudly than the systolic and at lower levels When the systolic murmur fails, actual timing of the murmur becomes quite essential to diagnosis, for its character will not betray its position in the cycle In timing listen to the sounds while a finger rests on the cardiac impulse

The diastolic murmur in cases of suspected aortic regurgitation *should be sought* for diligently along both borders of the sternum, and especially along the left border, where it is particularly apt to appear in cases of slight regurgitation that do not affect the pulse It should be sought in the lying and erect posture, and particularly with the breath held in deep expiration while the patient leans forward Even so in some cases it will prove elusive, vanishing completely from time to time

The degree of incompetence cannot be gauged either from the

loudness or the length of the murmur When the pulse indicates

unreliable, in gauging the state of the aortic valve It is the pulse that most reliably indicates the degree of regurgitation

A diastolic murmur is sometimes heard over the main artery of a limb in cases of conspicuous aortic incompetence, it is produced beneath the stethoscope and is not heard except when other signs of aortic reflux are quite obvious, consequently it has little value

DIFFERENTIAL DIAGNOSIS, INCLUDING PULMONARY REGURGITATION

The pulse —The characteristic signs found in the arterial system in aortic incompetence are repeated fully and usually in only one condition, namely, arteriovenous anastomosis Here the leak is from artery to vein rather than from artery to heart Every arterial sign of aortic reflux that has been named is repeated in free arterio-venous anastomosis and repeated in its extreme form The pulse has abruptness, though in less degree, in some cases of patency of the ductus arteriosus, where the leak is from aorta to pulmonary artery, it is also abrupt, in conditions of simple peripheral vaso dilatation, it may be full in exophthalmic goitre Aortic reflux, arteriovenous anastomosis, and patent ductus arteriosus (page 267) are distinguished by the signs that occur directly over the corre sponding lesions

The murmur —A low pitched blowing early diastolic murmur in the apical region strictly confined to the impulse in a case of auricular fibrillation with slow pulse is due to mitral stenosis (see page 145)

An early diastolic murmur heard in the 3rd and 4th left spaces in cases of mitral stenosis should be attributed, even when the pulse is unaltered, not to pulmonary, but to aortic incompetence The murmur described by Steell, and attributed to pulmonary re gurgitation consequent upon pulmonary hypertension in mitral stenosis, should never be diagnosed without X ray evidence of a dilated pulmonary artery The pulmonary valve is very rarely de formed except by congenital disease (page 271) or by acute bacterial endocarditis (page 194) When infection is certain and there are no arterial signs of aortic disease or of systemic embolism, a diastolic murmur at the pulmonary cartilage will sometimes render pulmon ary vegetations suspect

The to and fro murmur of aortic valve disease is sometimes diffi-cult to distinguish from to and fro friction sounds (described on

page 177) when these are confined to the base of the heart Difficulty in diagnosis occurs in cases of rheumatic carditis when a soft to and fro murmur develops in this situation and as yet other signs of pericarditis are obscure The point resolves itself with time There may also be difficulty in the same infection when widespread friction sounds have developed and, clearing away, leave a persistent to and fro murmur at the base, which may be interpreted either as pericardial or aortic in origin The presence of much regurgitation will be recognised or excluded by the pulse Uncertainty as to the presence of slight aortic valve damage, when the heart is known to have been exposed as a whole to rheumatic inflammation is of little consequence

RUPTURED AORTIC CUSP

Spontaneous rupture of a healthy aortic cusp does not occur Most instances of rupture are of cusps that have been affected by syphilis or subacute bacterial endocarditis a little aneurysm forms in the centre of a cusp and projects towards the ventricle one day this aneurysm breaks or a diseased cusp is torn from its edge to its base The accident is most likely to occur when the subject is making a considerable effort thus raising aortic pressure In such a case the subject may feel as though something has broken in the chest he becomes very breathless or falls in a state of collapse or actual unconsciousness and on occasion shows signs of acute dilatation of the heart Sometimes from the moment of the accident, sometimes a little later, the patient may notice a rhythmic noise within his own chest this may be the only symptom On examination the Wassermann reaction is generally found to be positive, and signs of regurgitation, usually free are discovered The diastolic murmur may be so loud that it can be heard by the ear placed near to the chest wall, it is often musical in quality (Fig 25), and is associated with thrill A similar murmur may occur in syphilitic aortitis when the cusp is retroverted

CONSECUTIVE PHENOMENA

Rapid pulse —In describing the signs of aortic regurgitation certain phenomena have been omitted because they are not the direct and immediate consequences of regurgitation, though they often follow from it As already said a chief consequence of free aortic regurgitation is a low mean blood pressure, the low pressure is the usual cause of the increased pulse rate that is common in this state, though it may also arise from infection

Vasodilatation —A phenomenon usual in free aortic regurgitation is vasodilatation, exhibited especially in the face, also in the upper limbs, and sometimes more generally The facial skin is of a fresh colour, is a little, or sometimes greatly, flushed, it is warm or hot The idea that aortic regurgitation causes pallor was erroneous it arose at a time when subacute infective endocarditis was unrecognised, for the latter disease affects the aortic valve and often causes intense pallor (pages 189 and 192), so does long continued rheumatism It is mainly because of associated vasodilatation that capillary pulsation (page 51) is so frequent in free aortic regurgitation, this pulsation is usually to be seen occurring spontaneously in the cheeks and forehead and can be elicited readily in the lip and lobe of the ear by



FIG 25 —Simultaneous electrocardiogram and a heart-sound record from the 2nd right intercostal space in a patient thought to have ruptured an aortic valve-cusp The murmur (*M*), which was early diastolic and musical, gives a record characterised by uniformity of the vibrations these occur at a rate of 133 per second The black rectangle marks the limits of ventricular systole 1st and 2nd sounds are indicated. Time in $\frac{1}{5}$ sec

gently pressing a slide upon these parts it is also seen though less frequently and conspicuously in the fingers It is only to be found in parts of the skin that are warm or hot and never in those that are cold, thus it is clearly linked with relaxation of arterioles Doubtless the large pulse pressure helps to render it conspicuous, but large pulse pressure is not its cause Capillary pulsation is seen equally well in cases of free union between the superficial femoral artery and vein, here too there are both high pulse pressure and the signs of vasodilatation If the common femoral vessel is obliterated by pressure and the arteriovenous anastomosis thus cut off from the general circulation, the pulse at once loses its extreme water hammer quality, the pulse pressure at once declines, but capillary pulsation remains of [illegible]

Anginal pain occurs in many cases showing aortic regurgitation it might be just to regard this pain as determined in some cases by the extra work that the valve defect imposes, but its inclusion in the symptom complex of aortic incompetence would not be in the best interest either of simplicity or clarity.

Aortic Stenosis

It is a sound rule rarely to diagnose conditions that occur rarely Aortic stenosis is far less common than aortic incompetence and its diagnosis should be infrequent It occurs chiefly in elderly subjects, occasionally in the rheumatic hearts of younger subjects

SYMPTOMS, SIGNS AND DIAGNOSIS

There are no symptoms of aortic stenosis

Anacrotic pulse —The outstanding diagnostic sign of aortic stenosis is a small pulse, rising slowly to a delayed summit, the condition should never be diagnosed *without this sign An experienced finger* will detect it, so often will inspection of pulsating arteries Sometimes an earlier summit can be detected on the slow upstroke by palpation, but more often in sphygmographic records (Fig 24), the pulse is then termed "anacrotic" This change in the pulse's form is the direct consequence of stenosis, the degree of change is related to the degree of stenosis It is for this reason that the sign is so essential, for, if the pulse rises quickly, such stenosis as may be present is not worth diagnosing The slow rising, and the anacrotic, pulse may occur in any other condition in which there is an incomplete and local obstruction anywhere between the aortic valve and the point on an artery at which the pulse is felt The mechanism is always the same—the primary wave of the pulse is spent in overcoming the obstruction, for this reason systolic pressure is lower than it otherwise would be No other lesion, however, produces this pulse with anything like the same frequency as does aortic stenosis In aortic aneurysm, through clot or through constriction of the mouths of branches arising from the sac, obstruction may occur in *outgoing* vessels The obstruction, however, is usually confined to one, less frequently to two, of the three vessels that issue from the aorta, *namely*, innominate, carotid, and subclavian artery so it is not very common to find the pulse universally slow rising in aneurysm, or to find it rising with equal slowness in the two arms A universal anacrotic pulse occurs as a rare phenomenon in cases of advanced

arterial disease with a weakly acting heart In all the different conditions named, including aortic stenosis, the systolic blood pressure, like the peak of the pulse, is reduced in level

Systolic thrill —The second sign is a systolic thrill at the base of the heart, usually maximal at the 2nd right cartilage or space This sign is usually to be detected if carefully sought, by placing the palm of the warm hand freshly on the base as the subject is completing a deep expiration to hold respiration in this phase It is sometimes felt most easily when the patient leans forward Though it is an expected sign, diagnosis may proceed without it, but a diagnosis cannot be made by means of it alone It can occur in aortic valve disease when there is little or no stenosis, and occasionally when no disease of the valve, but anaemia, is present

Systolic murmur —Every case of aortic stenosis presents a systolic murmur, heard loudest where the thrill is best felt and carried up to the clavicle or beyond into the neck along the carotid vessel A harsh murmur is the hint to examine for thrill The systolic murmur is a guiding sign but has no further value in diagnosis A systolic murmur at the aortic cartilage is usual in any form of aortic valve disease, in dilatation or aneurysm of the ascending aorta, in atheroma of the base of the aorta it is quite common in anaemia and in any condition in which the action of the heart is augmented To diagnose stenosis on the basis of such a murmur is unpardonable

X-ray —In the aortic stenosis of the elderly, the calcareous mass at the base of the aorta can usually be seen or photographed

Presence of aortic regurgitation —We have seen that, while the slow rising pulse is an essential sign, it is insufficient for the diagnosis of stenosis The systolic thrill or a harsh murmur strengthens the diagnosis by locating the obstruction But the most valuable localising sign is a diastolic murmur, for this indicates disease of the aortic valve Aortic stenosis rarely occurs without regurgitation, and so the former should rarely be diagnosed without simultaneous detection of the latter, but regurgitation must in this circumstance be diagnosed on murmur only, for an abruptly rising pulse is evidence against material stenosis

CHAPTER XV

DISEASE OF THE AURICULO VENTRICULAR VALVES

Pathological Anatomy

Mitral valve —Disease of the mitral valve usually results from an inflammation of the valve occurring during the course of active rheumatic disease (page 199) In the early phases of its inflammation, the two cusps present narrow bands of small sessile and closely set vegetations along their apposed surfaces Beneath and in the substance of the cusp is a more widespread inflammation The inflammation leaves, in healing a thickened margin to the cusp, the thin and flaccid edge, which is so necessary to perfect closure being destroyed Early thickening and rounding of the valve margin is a recognised cause of mitral regurgitation Repeated or long continued inflammation leads to higher grades of valvular deformity, the margins become more thickened and adhere together at their adjacent borders, at first the finest subdivisions of the chordae tendineae, later coarser subdivisions become matted together and to the cusp against which they lie The process spreads down the main chordae and these and the cusps become cemented and encased in new tissue Gradually the valve apparatus is converted into a contracted and rigid structure, and pierced by a buttonhole or funnel shaped orifice that can neither close nor open This is stenosis The rheumatic inflammation that first produces crumpling with regurgitation ultimately produces stenosis, it is one disease presenting a varying picture at different stages of its progress

There is a condition of atherosclerosis of the cusps, and especially of the aortic cusp of the mitral valve, that sometimes proceeds to calcareous deposit sufficient to cause stenosis Calcareous valves in elderly subjects are sometimes of this kind, they may be formed earlier by deposition of lime salts in tissues sclerosed by rheumatism Syphilis does not damage the mitral valve

In enlargement of the left ventricle it is frequent to find the

chamber and auriculo ventricular ring dilated after death and the whole mitral valve enlarged, its chordae long, the valve curtains having a wider sweep, but their substance thicker and more opaque than normal Such valves are probably often incompetent, we have, however, no adequate test of mitral incompetence at autopsy

Infective endocarditis ulcerates the valves and often cuts the chordae, thereby rendering the valve incompetent, or by heaping up vegetation may cause obstruction at the orifice

Tricuspid valve —The only common cause of lesions of the valve is rheumatism, and it follows, just as does mitral disease, from a characteristic inflammation of the valve The tricuspid, however, is less frequently and far less severely involved than is the mitral valve When stenosis is present it is slight, the thickened valve edge forming a fixed ring of considerable size, severe mitral stenosis usually coexists

Mitral Stenosis

ITS RECOGNITION

Although many symptoms may be complained of by patients suffering from mitral stenosis, there are none that can be ascribed properly and usefully to this deformity of the valve A number of patients with developed stenosis are quite free from symptoms, and the discovery of the disease comes as a surprise Only signs of the disease will be discussed here

An abrupt and accentuated 1st sound at the impulse, an accentuated or reduplicated 2nd sound at the pulmonary cartilage, are signs commonly occurring in mitral stenosis, but these signs alone or in combination, with or without an associated history of rheumatic fever, form an insufficient basis for diagnosis They result oftener from [illegible] t

obtain The presence of signs supposedly indicating hypertrophy of the right ventricle should not be cited as evidence of mitral stenosis, this method of procedure is too indirect to be sound One sign only justifies the diagnosis, and that is the appropriate murmur The corresponding thrill, when this is present, is of course to be included, it is no separate phenomenon, the ear hears and the hand feels much the same vibrations when these are both audible and palpable The murmur of mitral stenosis is the only heart murmur that requires much study.

A word first of all about the thrill It is best felt with the palm of the hand freshly applied to the chest wall in the region of the impulse, to have value it should be sufficiently sustained to possess a distinct purring quality It is unwise to accept as thrill anything but an unmistakable sign The thrill has much the same distribution in the cycle as the murmur, though it is not always perceived to last so long it is more elusive than the murmur and less important in diagnosis

The murmur is almost always loudest at the impulse or close to it, and is usually confined closely to this region, though sometimes heard over a wider area of the precordium It will be described first as it is derived from a normally beating heart, and, later, the sounds as modified by auricular fibrillation and other abnormal mechanisms will be discussed The description, which in certain aspects differs from earlier teaching is based upon a long experience of auscultation and, perhaps uniquely, upon a close comparison of what is to be heard with what is to be recorded by microphonic methods in a large number of selected cases The graphic method alone places the time relations of these murmurs beyond dispute

With normal rhythm —The characteristic murmur is of low pitch, usually rough rumbling or rasping and ends abruptly in a loud sound with which it is continuous This sound is in fact the first sound of the heart, which being usually greatly accentuated is chiefly responsible for the idea that the murmur rises in a crescendo It is erroneous to believe that the murmur is usually recognised by timing it, although this is steadily taught Most people, though capable of identifying the chief murmurs readily, cannot and never will time murmurs reliably They recognise this murmur of mitral stenosis the instant it is heard, as I do myself in routine work, by its low pitched abruptly ending noise Much labour is lost and many ultimately fail to know this murmur through persisting in the effort to time, instead of learning to know it as one learns to know a dog's bark

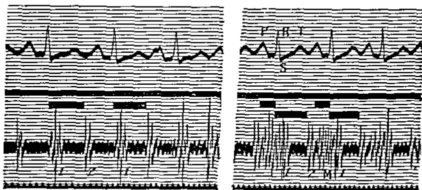
The murmur is due to the passage of blood through the constricted orifice of the mitral valve in diastole The phases of diastole in which the murmur is audible are those during which the blood flows at an adequate rate through the mitral ring The rate of flow, adequate to throw by means of eddies the structures involved into vibration, may be attained for a shorter or longer part of diastole What parts of diastole are filled by murmur depends mainly upon the rate of heart beat and upon the degree of stenosis Given that a certain

amount of blood has to flow through the orifice each minute, the velocity of flow will be greater as diastole becomes relatively shorter with faster heart rate, in like circumstance the velocity will be greater as the orifice is smaller In most cases of mitral stenosis, as these come under examination, the rate of beating is rapid and the stenosis is moderate or considerable, consequently the velocity of flow is high and it is *the rule* for the murmur to occupy all diastole, beginning with the 2nd sound and ending with the abrupt 1st sound (Figs 26, 27, and 28) Names become stereotyped, and it is still customary to hear this full diastolic murmur called presystolic, although this term can rarely be used with accuracy When the heart is beating at 120 per minute, as it often does in out patient clinics, it clearly cannot be used accurately, for in these patients diastole is no more than $\frac{1}{4}$ second and presystole, shall we say, $\frac{1}{12}$ second in duration The point would not be stressed so much were it not important to realise that there is a manifest tendency, which is traditional, for the medical profession to exaggerate the accuracy of its subjective methods of examination In using any method of mensuration it is essential to know the error of it, as has been stated earlier in connection with the measure of cardiac enlargement (page 121), and the error in timing can scarcely be less, it is usually more than $\frac{1}{10}$ second Graphic registration, and even very attentive auscultation, directed especially to detect a gap between 2nd sound and murmur, shows that murmurs confined to late diastole are comparatively rare They are only displayed by hearts that beat slowly and in which the stenosis is not advanced In rare cases in which

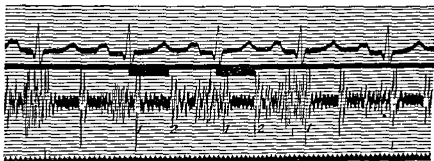
of ten years At this stage, and for a few years, the murmur characterising mitral stenosis in these young subjects very often occurs in mid diastole (page 203) The reason for its curious position is imperfectly understood, but it is well to know that this variation occurs

Early diagnosis—In teaching that the appropriate murmur is the only sign justifying a diagnosis of mitral stenosis, I do so subject to the reservation that the murmur is diligently sought, and that reasonable effort has been made to elicit it The murmur is heard with much greater frequence when the patient lies than when he stands, and oftener when the patient lies upon the left side than upon his back It is heard with especial frequency when one or other of these two last positions has just been assumed by a patient

who has been sitting, or has just taken sufficient exercise to raise the pulse rate and the velocity of blood flow through the orifice, as in sitting up and lying down a few times Close *attention should* be paid to the sounds at the impulse during the first 5 or 10 beats



FIGS 26 and 27 —Simultaneous electrocardiogram and sound record from a case of mitral stenosis. The first sound record (Fig 26) was from a point a little internal to the impulse and shows accentuated ... *taken from the impulse at which*



... from a case of mitral stenosis ... a diastolic rumble and between 2nd and 1st of ventricular systole

of the heart immediately following the assumption of one or other of these postures after exercise *So often is the murmur audible in* these circumstances only, and for a few cycles only, or so often does the murmur become unmistakable only under these same conditions, that no subject deemed likely to have mitral stenosis should be proclaimed free of it until these special tests have been employed They

should be used in every case of suspected heart trouble where there is an accentuation or a reduplication of the 1st sound, or of the 2nd pulmonary sound, or where there is an apical systolic murmur If they are so used as a routine, experience soon shows that the signs just named are useful guides in suggesting the special tests, but once so used, can be neglected as actual diagnostic signs of mitral stenosis Under the routine here described, which eventually rejects all signs but the appropriate murmur, early mitral stenosis will very rarely be overlooked What is of equal, if not of greater, con sequence, is that there will be little likelihood of diagnosing this disease of the valve where it does not exist There are, of course, cases in which, owing to the presence of venous congestion, exercise or postural tests are undesirable, it is of little consequence to such patients whether a diagnosis of mitral stenosis is made or not

In auricular fibrillation —When the auricles are fibrillating, the character of the murmur of mitral stenosis does not usually differ from the common murmur heard while the rhythm is normal That is to say, it is a low pitched, rumbling, or rasping murmur In most untreated cases, seen for the first time, the ventricles are beating rapidly In such circumstances the murmur occupies full diastole and ends in an abrupt 1st sound, thus giving as usual a crescendo effect

In cases of normal rhythm, as we have seen, the murmur usually occupies full diastole, but with slow heart action may become confined to one part of diastole, the favoured part of the cycle is then presystole In auricular fibrillation when the ventricle slows, the murmur likewise fails to occupy full diastole, but the favoured part of the cycle is now early diastole That is so because the auricles have ceased to function and to force blood rapidly through the orifice at the end of diastole, and in these circumstances the entry of blood from the auricle is at its fastest immediately after the opening of the auriculo ventricular valves, the auricles being then engorged with blood that has been dammed up in them during ventricular systole The velocity soon lessens as diastole proceeds, and so the murmur in question tends in general to be diminuendo In cases of fibrillation where the ventricular action is very slow, the murmur is confined to early diastole in all cycles and, for an unexplained reason, often tends to lose its rasping quality, becoming soft and resembling more in character the murmur of aortic regurgitation Usually the ventricle beats less slowly though irregularly, and the murmur retains its characteristic roughness, it fills the whole diastole of short cycles, and fills early and mid diastole in long cycles, tailing away and leav-

ing an appreciable pause between the end of the murmur and the subsequent 1st sound This combination of sounds is frequent and should be known thoroughly, it requires a little thought to understand it, to help its appreciation I have inserted a graphic record of such sounds taken simultaneously with an electrocardiogram (Fig 29)

When auricular fibrillation develops in cases of mitral stenosis with normal rhythm, a late diastolic murmur previously heard sometimes disappears or becomes much less distinct owing to the loss of auricular systole It is well recognised that in auricular fibrillation,

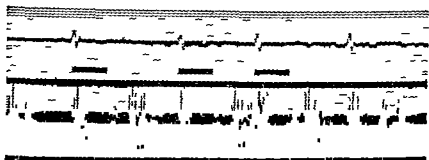


FIG 29 —Simultaneous electrocardiographic and heart sound record from the region of the apex in a case of mitral stenosis and auricular fibrillation under digitalis Each systole of the ventricle the limits of which are indicated by the black rectangles is accompanied by 1st and 2nd sound Shortly after the ending of the 2nd sound a coarse diastolic murmur (M) appears When diastole is long this murmur is diminuendo and ends in diastole but when diastole is short as in the second cycle it ends in the next 1st heart sound Time in $\frac{1}{10}$ sec

mitral stenosis may be concealed, the heart at times presenting no murmurs The tests previously described will usually elicit the murmur, or render a doubtful murmur unmistakable, here, as in instances of normal rhythm

developed	diastolic	
3 Rate rapid, stenosis developed	Full diastolic	Full diastolic
4 Rate moderate, stenosis developed	Full diastolic	Full diastolic in short cycles, early and mid diastolic in long cycles

This table shows at a glance the part played by auricular systole in determining the phase of diastole in which the murmur appears

The influence of auricular systole can also be studied in cases of mitral stenosis associated with heart block If the interval between auricular and ventricular contractions widens, a shorter or longer gap of silence is noticeable between the murmur and the 1st sound If 2 1 block develops, then two similar murmurs will occur, the one in presystole and the other in early diastole, and each the result of a corresponding beat of the auricle Instances of these kinds are not very uncommon, instances of mitral stenosis and complete block in which the murmur alters its position from cycle to cycle are rare

Flint's murmur —This murmur may be defined as one that resembles the murmur of mitral stenosis, and is heard in cases of free aortic regurgitation in which mitral stenosis is subsequently proved absent The precise manner in which the murmur is produced is unknown, it is not uncommon Very rarely it may be as rasping as that of mitral stenosis and may, so it is said, be accompanied by a thrill, usually it is of low pitch, is rumbling in quality, occupies full diastole, but is less audible than the similar murmur of stenosis In cases of aortic regurgitation, where a diastolic rumble is heard at the impulse, it is impossible to tell the origin of the murmur from its character or timing alone Those who aim at complete anatomical accuracy in diagnosis will come nearest to accomplishing their purpose by using associated information If regurgitation is slight, if the heart is small, or there is a history of rheumatic fever or chorea, mitral stenos s may be d [illegible]

the decision taken one way or the other will alter neither prognosis nor treatment

Mitral Regurgitation

SYSTOLIC APICAL MURMURS

Cardiorespiratory murmur —This is the commonest murmur heard in the region of the heart's impulse It is usually heard best at or a little beyond the impulse Often it is audible over a wide area, being frequently distinct not only over the precordium but in the axilla and at the angl of th l ft [illegible]

broken into two or more short murmurs by the heart beats But until thoroughly known its respiratory relationship is generally overlooked, when a short and inconstant murmur of superficial quality is heard, its relationship to respiration, and the effect of suspended breathing upon it, should be observed The ventricle at each systole creates a little vacuum in its neighbourhood and so increases the rate at which air enters the lung It is a normal phenomenon when the heart is beating quickly and strongly, as in exercise or excitement, in steep uphill walking, when the mouth is open, a sharp movement of the air past the larynx, at each systole of the heart, can often be appreciated by the subject The murmur should be construed as the natural accompaniment of the overacting heart

Exocardial murmurs —Precordial systolic murmurs that are short, seem superficial, and have any scraping quality, are usually regarded as produced outside the heart by roughening of pericardium or pleura, the opportunity of examining the suspected membrane does not often occur When it does occur, it is sometimes, but by no means always, possible to find in them a lesion to which the murmur may be attributed, proof, however, is rarely forthcoming During life the distinction between systolic murmurs that arise within and without the heart, so called "endocardial' and "exocardial" murmurs, is often impossible, unless the latter are represented by the characteristic, widely heard, to and fro friction sounds of recent pericarditis, or are frankly cardiorespiratory

Inconstant murmurs —Not infrequently a soft, blowing systolic murmur is heard in the region of the cardiac impulse while the patient is in one posture only, usually the lying, sometimes the standing Some murmurs of the same character vary much in intensity from day to day, others appear only after exercise Such murmurs are usually audible over a relatively small area of chest wall, though this is not always so

Constant murmurs —A considerable proportion of systolic apical murmurs show relative constancy from cycle to cycle and from day to day, these are in general the harsher and longer murmurs, and are heard over a wider area of precordium and axilla than the inconstant murmurs previously described They are the murmurs that are usually regarded as chiefly signifying mitral regurgitation Among them is the murmur that starts in mid systole, a delay which causes it frequently to be mistaken for one occurring in early diastole.

MITRAL REGURGITATION AND ITS SIGN

It is desirable to emphasise at once that in discussing the diagnosis of mitral regurgitation we are not discussing, except indirectly, the diagnosis of mitral disease, the two problems must be dealt with separately

There are no symptoms to be attributed usefully to mitral regurgitation The only sign that can be named is a systolic murmur It has been said that in mitral regurgitation the 1st sound of the heart may be replaced by murmur, graphic records always show the 1st sound The fact is that the ear in listening for the 1st sound is confused by a loud murmur

A real difficulty in attempting accurately to correlate certain systolic murmurs with mitral regurgitation lies in the frequent impossibility of stating, from the appearance of the valve after death, whether leakage has occurred or not during life From this standpoint it may be said that if regurgitation can be declared to have happened in any one group of cases more than another, it is in the group in which the cusps are subsequently found so thickened that the rigid valve is incapable either of opening farther or of closing During life these cases of stenosis should present the sign of regurgitation The facts are these In about one third of the cases no murmur can be discovered in the remainder a systolic murmur is heard in the region of the impulse This murmur may be quite local, more often it may be followed well out to the axilla It may be soft almost to inaudibility, it may be musical, or it may be harsh and loud, occasionally it may be associated with thrill It is usually constant from cycle to cycle and from time to time, but is not always so Thus the sign of regurgitation is to be recognised as very variable from case to case Moreover, all these varieties of murmur are heard from time to time in cases in which at autopsy no real fault can be found with the valvular apparatus It is then quite usual to suppose that the muscular ring surrounding the base of the valve is relaxed sufficiently to produce leakage In the past we have been near to believing on *a priori* grounds that a distinct systolic murmur at the impulse must be caused by regurgitation, identifying a systolic murmur and then assuming it to arise out of regurgitation In most instances this assumption may be right, in some instances it is wrong, for there are other ways in which these murmurs are produced There are very definite elements of weakness in our way of diagnosing mitral regurgitation It is never very satis

factory to base a diagnosis upon a single sign, and the position is insecure when the sign is known not to be of a high grade of reliability

If we are treating a case of acute infectious disease, or a frail elderly person, and we are sure in such that a systolic murmur has recently developed at or near the impulse, we may be justified in interpreting this as the first indication that the heart is dilating, and so it may be important, but we are hardly justified in definitely drawing this conclusion unless, simultaneously or later, direct signs of enlargement can be found The systolic murmur is chiefly a guiding sign, and as such may have a distinct value An open window may be such a sign to a police officer on night rounds, he may suspect, but does not necessarily conclude, there is a thief in the house A fixed systolic murmur in cases of chronic heart affection has a similar value in that it is sometimes the first sign to attract attention and lead to close examination of the heart

Confidence in the value of a diagnosis of mitral regurgitation is reduced not only by frequent doubts as to whether regurgitation actually exists or not, but by our total inability to state its degree if it is there There may be some grounds, though they remain inadequate, for claiming that harshness, thrill, audibility over a wide area, or other characteristics, mark the murmur of mitral regurgitation, no claim has yet been made that particular qualities betray the slight or the large leak Assuredly if the diagnosis of regurgitation, *qua* regurgitation, matters, it matters according to its degree

Briefly, the diagnosis of mitral regurgitation has a very limited importance, it may be useful as an early indication of muscular failure in acute infectious disease and occasionally in old age Further reasons will be given for concluding that it should not be allowed to modify either prognosis or treatment (page 156)

Recognition of Mitral Disease

To recognise mitral disease by means of systolic murmurs, it is *first* essential to be accurate in eliminating all murmurs, cardio-respiratory or not, that arise otherwise than from the mitral valve, it is secondly essential to differentiate clearly between a mitral murmur that is due to faulty ventricle or to diseased valve To regard any distinct systolic apical murmur as meaning a diseased valve is indefensible The statement that a constant, long, harsh systolic

murmur heard at or beyond the impulse *usually* signifies mitral disease, and that this interpretation is more reliable when there is a history of rheumatic fever, is as strong a statement as can be defended

Accurate diagnosis of disease of the mitral valve depends upon the recognition, not of mitral regurgitation but of mitral obstruction It has been stated already that rheumatic fever produces both these defects and that the underlying conditions are but stages of one and the same disease Thus diagnosis of rheumatic mitral disease is accomplished most accurately (and in speaking of

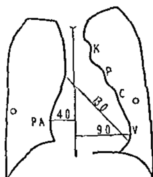


FIG 30 —Orthodiagram Man aged 24 weight 113 lb Mitral stenosis (developed) Below the aortic knob (*K*) appear the projection of *P* the dilated pulmonary artery of *C* the enlarged conus and the outline of the ventricle (*V*) the apex of which is unusually round The right auricular shadow (*RA*) is enlarged

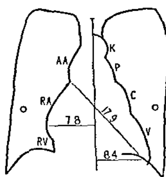


FIG 31 —Orthodiagram Man aged 29 Mitral stenosis (developed) aortic regurgitation (slight) and great enlargement of the heart The right border is composed of ascending aorta (*AA*) which is displaced an enlarged right auricle (*RA*) and the beginning of the enlarged right ventricle or the vena cava (*RV*) The left border consists of aortic knob (*K*) dilated pulmonary artery (*P*) greatly enlarged conus (*C*) and ventricle (*V*)

accuracy we must take into account not only correct guesses but the elimination of incorrect guesses) at the present time by starting at stenosis and carrying back the diagnosis of this lesion as far as is possible If the method of early diagnosis (page 143) is followed, many cases that might otherwise pass as mitral regurgitation are transferred into the decisive category of stenosis, by using care to unveil these early cases, the attempt to interpret many systolic murmurs becomes needless Of the cases that remain it is questionable if a diagnosis of mitral disease can be made accurately in a sufficient percentage to warrant the attempt upon the basis of present physical signs

Tricuspid Affections

Tricuspid stenosis —There is only one sign of this condition that has much value it is a murmur like that described for mitral stenosis, but occurring over the lower part of the sternum Characteristically in these cases, a murmur of mitral stenosis is heard at the impulse, is lost, and the similar murmur of tricuspid stenosis becomes audible as the stethoscope is moved inwards towards and to the sternum Absence of a tricuspid murmur does not mean that the valve is undiseased Large pulsation of veins and liver is common The diagnosis should be confined to cases of mitral stenosis, when made it does not affect the management of the case

Tricuspid regurgitation —A constant blowing systolic murmur, confined to the region of the lower sternum and ensiform cartilage, is not uncommon in cases of venous congestion and is usually ascribed to tricuspid regurgitation Much more often than not, in cases of general venous congestion, no distinct murmur of the kind can be heard, moreover, a similar murmur is audible from time to time, and especially after exercise, in people who are apparently in good health It is probable that the tricuspid valve readily becomes leaky

In the full hypothesis of progressively marching back pressure, the pressure was supposed to rise in the right ventricle before it rose in the veins, and the tricuspid valve was pictured as one of the last lines of defence Incompetence of this valve was regarded as the signal for engorgement of the veins and so tricuspid incompetence came to be used almost as an equivalent term for venous engorgement The hypothesis neglected the facts that, by obstructing the outlet from the right ventricle, this chamber cannot be engorged without raising venous pressure correspondingly, moreover, the rise of pressure in venous congestion does not take place abruptly, as it would under this theory, but gradually over a period of hours or days Thus venous [illegible] not begin in a sudden leakage of the tricuspid valve [illegible] ipetence by [illegible] l incompetence, the diagnosis is in general merely a statement of the discovery of a murmur, which is then assumed, perhaps in many cases rightly, to indicate this regurgitation The degree of reflux is not indicated, the influence of the reflux cannot be estimated unless it can be detected in deformity of the waves in the cervical veins, and this is doubtful

Tricuspid incompetence forms an instructive example of an old-time clinical philosophy An intangible valvular defect, draped with complex but unproved hypothesis, has formed a screen, tending to conceal the essential The essential is not tricuspid incompetence, but general venous congestion, and the precise information that is required for practical purposes is obtained, not from the precordium, but from the veins themselves, for in this system lie the early, as well as the late clinical signs of congestive failure The veins provide direct signs, and those thoroughly familiar with them use neither the tricuspid murmur nor the theory that goes with it

who have outlived for some years the period of responsible infection, there are some who display aortic regurgitation or mitral stenosis, but in whom bodily vigour is unimpaired and the cardiac reserves are seemingly intact, in whom there is no complaint of symptoms, in whom no very definite signs of cardiac enlargement can be found They are usually, but not exclusively, young subjects, and experience shows that in them the prognosis is in general good, that is to say, these people will usually live many years, and enjoy good health In this statement lies the basis of correct prognosis in so far as the valve disease is concerned, but it must here be emphasised that in speaking of valve disease I am speaking purely of aortic disease or of mitral stenosis, and am not taking into account any of the common accompaniments of these diseases A few in this uncomplicated group of aortic disease and mitral stenosis, and a few only, will develop graver manifestations in a few years It is most important, however, that this fact should not be allowed to introduce gloom into the prognosis of the group as a whole It is right and proper that the general attitude to the group considered should be one of almost unqualified optimism, provided that the subjects individually realise the wisdom of simple precautionary measures In this group there is rarely the means of discriminating between the many who will make good and the few who will fail, therefore the attitude, to be consistent, must be optimistic to all or pessimistic to all If it is optimistic to all, it may be that an occasional patient will fail without much warning, and perhaps dramatically, but the majority of the patients will enjoy what they have a right to enjoy, namely, a reasonable sense of security If it is to be pessimistic to all, most of them will needlessly experience periods of apprehension or alarm and a constant feeling of insecurity Surely there can be but one decision in considering these alternatives, for the first only is reasonable, or indeed humane

If what has been said has been understood, then it is unnecessary to proceed to discuss the case of valve disease that presents minor

focus of discussion, namely, mitral regurgitation This will be discussed before summing up

Mitral regurgitation —The difficulties inherent in the diagnosis of mitral regurgitation have been discussed already, it has been shown that the diagnosis is insecure, and that when regurgitation seems

certain its degree cannot be stated Since the diagnosis, when made, hangs exclusively upon one sign, there is no advantage in discussing the significance of mitral regurgitation as such It is more rational in the circumstances as far as possible to escape hypotheses which deal with the effects of regurgitation, etc , and to deal with the plain facts, asking what is the prognostic significance of a systolic apical murmur, and, if it is considered desirable, distinguishing this murmur by certain characteristics, such as length, harshness, and conduction We are considering a murmur that is constant, and not a murmur that has just appeared (see page 150). Now, if we take the first fifty subjects encountered who present such a murmur and the first fifty who do not, there is no doubt that the prognosis in the first group will be less satisfactory than in the second But it is beside the point so to discuss the matter, for the second will not form a justifiable control group to the first It is quite essential, if the significance of the apical systolic murmur is to be ascertained, that the two series of cases should be precisely alike in every respect apart from the murmur The second group, therefore, must include an equal number of subjects presenting breathlessness, a history of rheumatic fever, etc It never has been shown by such comparison that there is any material difference in the prospects of the cases in these two groups Actually, when such comparisons have been made, no differences have been ascertainable Beliefs that there is a material difference are based upon theoretical assumptions So firmly fixed is the traditional view, however that the critical method of examination and its result are almost always evaded Thus if it is stated that mitral regurgitation has little or no prognostic significance, the question is not infrequently asked if it has no significance when associated with ventricular dilatation or recent rheumatism The reply is that significance attaches to dilatation and to recent rheumatism

The line of argument here employed is largely a repetition of that already used in discussing the significance of extrasystole The principle at stake, however, is so vital to prognosis in general that this re-emphasis is expedient An example from a different field will serve to establish the point that associated circumstances are really irrelevant A simple healed scar on the tongue clearly has little or no prognostic significance, yet patients who display such scars have not a normal outlook, since some of them are epileptic The fact does not alter the prognostic significance of the scar itself

The prognosis in cases of aortic regurgitation has often been stated

who have outlived for some years the period of responsible infection, there are some who display aortic regurgitation or mitral stenosis but in whom bodily vigour is unimpaired and the cardiac reserves are seemingly intact, in whom there is no complaint of symptoms in whom no very definite signs of cardiac enlargement can be found They are usually, but not exclusively, young subjects, and experience shows that in them the prognosis is in general good, that is to say, these people will usually live many years, and enjoy good health In this statement lies the basis of correct prognosis in so far as the valve disease is concerned, but it must here be emphasised that in speaking of valve disease I am speaking purely of aortic disease or of mitral stenosis, and am not taking into account any of the common accompaniments of these diseases A few in this uncomplicated group of aortic disease and mitral stenosis, and a few only, will develop graver manifestations in a few years It is most important, however, that this fact should not be allowed to introduce gloom into the prognosis of the group as a whole It is right and proper that the general attitude to the group considered should be one of almost unqualified optimism, provided that the subjects individually realise the wisdom of simple precautionary measures In this group there is rarely the means of discriminating between the many who will make good and the few who will fail, therefore the attitude, to be consistent must be optimistic to all or pessimistic to all If it is optimistic to all it may be that an occasional patient will fail without much warning, and perhaps dramatically, but the majority of the patients will enjoy what they have a right to enjoy, namely, a reasonable sense of security If it is to be pessimistic to all, most of them will needlessly experience periods of apprehension or alarm and a constant feeling of insecurity Surely there can be but one decision in considering these alternatives, for the first only is reasonable or indeed humane

If what has been said has been understood, then it is unnecessary to proceed to discuss the case of valve disease that presents minor degrees of failure or of cardiac enlargement The general arguments that have been advanced, however, may be given further point by dealing with the example of valve defect that has been the chief focus of discussion namely mitral regurgitation This will be discussed before summing up

Mitral regurgitation —The difficulties inherent in the diagnosis of mitral regurgitation have been discussed already, it has been shown that the diagnosis is insecure, and that when regurgitation seems

origin, the aorta and parts of the ventricles have been exposed to spirochaetal infection, the disease is progressive, and there is considerable possibility of damage to the mouths of the coronary vessels A diagnosis of aortic regurgitation of syphilitic origin often acquires an added significance by extension of the diagnosis to neighbouring structures Properly speaking, these considerations are irrelevant to the prognosis of the valve disease itself, but they are certainly relevant to the question of the significance of valve disease in the more general sense It will be no great exaggeration to state that the greatest significance attaching to a diagnosis of valve disease is the light it often throws upon the history of the heart affected by it and the help it gives in leading up to a diagnosis of decreased reserve, of active infection, of disease of the aortic wall, or of coronary obstruction

Management

When active disease is present in the valves, the treatment concerns the infection, this is dealt with in so far as it is understood under the appropriate headings of bacterial endocarditis (pages 194 and 196), rheumatic endocarditis (page 205) and syphilitic aortitis (page 229) This really ends what has to be said of the treatment of valve disease, since chronic and inactive lesions can hardly be regarded as susceptible to treatment Surgical attempts to relieve cases of mitral stenosis presenting failure by cutting the valve have so far failed to give benefit I think they will continue to fail, not only because the interference is too drastic, but because the attempt is based upon what, usually at all events, is an erroneous idea, namely, that the valve is the chief source of trouble

The treatment of *patients* displaying valve defects will be found in other parts of this book, the treatment is not of the valve defect [illegible]

[illegible] to do so this attitude has been unwise Thus digitalis was at one time forbidden in cases of aortic regurgitation, under the plausible pharmacological plea that it must increase regurgitation by lengthening diastole and raising arterial pressure, but the moment the facts were investigated clinically it was found that digitalis produces neither of these effects in the aortic case The proper use of digitalis is governed by quite different considerations, such as the presence of auricular fibrillation, and these are not

different in aortic and other cases The valve deficiency may be said itself to affect treatment chiefly when the deficiency is uncomplicated If a patient has aortic regurgitation, aortic stenosis, or mitral stenosis, but presents no sign of cardiac enlargement, and has a good exercise tolerance, *it is a wise precaution to forbid any very* strenuous act of exercise or long hours of manual work To confine such cases to purely sedentary occupations and to warn them against all acts of exercise, even to forbid quick walking, cycling, swimming, is not to treat them well On the contrary, exercise well within the limits of tolerance is to be encouraged, only a limitation of bodily activity is to be insisted upon The same patients, having recognisable cardiac weakness, should be acquainted with and be asked to conform rigidly to simple rules of health (page 289).

The restrictions will naturally be increased to suit the needs of patients in whom the valve defect is complicated by associated conditions

When a patient presents what is regarded as uncomplicated mitral regurgitation, or, more strictly speaking, exhibits a persistent systolic apical murmur, the heart being of normal size and tolerance of exercise perfect, and there being no reason to suspect infection, it is my habit to leave bodily activity quite unrestricted and to encourage the full continuance of all pursuits, however vigorous these may be, to pass the patient as a recruit for military service, and to advise ordinary premiums for purposes of insurance This policy is not settled by any theoretical consideration, but its soundness has been proved by practical experience It naturally carries with it the proviso that he who adopts it must neither carelessly assess exercise tolerance, nor neglect to examine the heart scrupulously from other points of view To illustrate this much contended point, there is the case of a young man, seemingly possessing the perfect health and exercise capacity of an athlete He was sent to see me in 1922 because he had been found to have a blowing systolic murmur near the heart's apex conducted out toward the axilla Such a murmur was indeed present, but all other signs were quite natural He wished to climb Mount Everest and, with my consent, did so, going as high as any living man has climbed (over 27,000 feet) and returning and remaining since in good health This emphatic example is exceptional only in the unusual severity of the test imposed

CHAPTER XVII

HEART STRAIN, OVERWORK, AND FAILURE

Heart Strain

It has long been thought that the healthy heart frequently suffers strain as an immediate result of strenuous or prolonged bodily exercise, and this idea conforms with, and forms part of, the more general and long held conception that heart failure results mainly from the heart being overburdened by work

THE BURDEN

In discussing heart strain it is not always remembered that the strain of weight lifting or of other strenuous act never falls directly upon the heart, it falls immediately upon muscle tendon, and bone, the load on the heart is increased through the blood pressure The muscular effort may be abrupt and violent, the load falls on the heart more gradually and less severely as the circulation responds to a call set in motion through normal physiological channels The chief sources of increased energy expenditure by the heart in exercise come from increased rate of beating increased output, and raised blood pressure

ACUTE STRAIN DILATATION

When after some unusually strenuous or violent effort the muscle of a limb at once gives pain and is locally tender, but shows no gross damage, the reason may be that some of its fibres have been injured, the idea that a similar event happens in the case of a healthy heart muscle is not uncommonly held, this idea is unsupported by fact and overlooks what has just been stated namely, that the load placed on the muscle of the heart is a blood pressure load, and unlike the strain that a heavy weight places directly upon the somatic muscle

A more commonly held view is that the overloaded heart dilates

The most striking instance of what is to be interpreted as acute dilatation of the ventricle under a suddenly increased load is found in the occasional consequences of rupture of an aortic valve cusp The rupture happens usually during bodily effort, while blood pressure is raised, and the subject may become breathless, cyanotic, and perhaps unconscious There is no reason to doubt that in these circumstances the heart has become dilated and embarrassed, but the precise circumstances should be noted It may be assumed that the rupture occurs in early diastole, for that is the time at which the valves will be in greatest tension, at this phase of the cycle the aorta is full of blood and the pressure within it, as a consequence of bodily effort, is unusually high The regurgitant stream of blood in these circumstances must be large and is directed into a rapidly relaxing or relaxed ventricle The ventricle is submitted to a great reflux abruptly, without warning of any sort, and in a moment during which it is powerless to react or adjust itself The production of an acute overfilling in such circumstances is not difficult to understand, especially if we assume, as we are almost certainly justified in doing that the heart experiencing this accident and showing this response is generally, if not always, an unhealthy organ

In simple bodily effort the circumstances are quite different Here the heightened pressures of the arterial system are encountered by the ventricle only during its systole, these are quite insufficient to embarrass a healthy ventricle, which, however, will dilate in diastole to accomodate an increasing input of blood at a relatively very low pressure from the veins The increase of input is met by increase of the output of the heart It is now generally recognised that any increase in the size of the normal heart as the result of exercise is confined to the period of exercise itself and disappears promptly when exercise ends, for no enlargement is ever found once exercise is stopped, the size of the heart is less easily measured with exactitude during exercise, but evidence that change occurs during strenuous effort or prolonged exercise has been obtained by means of X rays Dilatation when observed during exercise has been slight, and we may be content with the statement that great dilatation does not occur normally The heart in these circumstances dilates within limits that are to be regarded as physiological A fuller diastole, with increase of the output per beat, has been shown to increase the efficiency of the organ as a pump Thus a dilatation in exercise would not be regarded by contemporary physiology as injurious *but* as distinctly advantageous

Moreover, we know that injurious dilatation, if such is ever threatened, is safeguarded by the parietal pericardium, a comparatively unyielding membrane limiting the capacity of the heart to dilate excessively in health There are abundant examples in which the healthy, or comparatively healthy, heart becomes greatly distended for shorter or longer periods without undesirable after-effects occurring A simple instance is where the heart is dilated by inspiring strongly with the glottis closed If this inspiration is maintained, the heart on the X ray screen is seen to dilate very greatly Yet this experiment can be performed again and again by healthy men with impunity In paroxysms of tachycardia, as has been described, *the patient's heart may become so dilated that the impulse* passes well out into the axilla, and the veins and liver may engorge acutely The attack, in which the heart is beating perhaps at 200 beats per minute, continues thus for many hours or days Yet, when the paroxysm ends, the heart resumes its original size, often after only a few beats, and within a short time the patient is as he was before the attack Such paroxysms, even if repeated, impose no recognisable injury on the heart, yet the dilatation is greater, and far more prolonged, than any that can reasonably be suspected from present evidence to occur in effort or prolonged exercise

The idea that the heart may be strained by a physiological act has received a good deal of stimulus from the incorrect interpretation of certain instances of acute dilatation of the heart, attributed to effort, or occurring during pregnancy All the cases of this kind that I have been called to see, and in which there were clear signs *of dilatation*, have been instances of *unrecognised* paroxysms of tachycardia, the mistake, however, is less frequent than formerly In the interpretation of these cases two erroneous ideas came into play The first was that strain can cause pathological dilatation The second was that dilatation of the heart causes tachycardia, actually dilatation of the heart has little influence upon its rate In these cases the tachycardia is primary and pathological and it causes the dilatation (pages 83 and 86)

Paroxysms of tachycardia in certain patients are provoked by exercise In such a patient the first attack complained of may have been determined by some act of exercise There has been a strong, perhaps a natural, tendency to ascribe the attack altogether to the act of exercise in this circumstance But close enquiry shows that this interpretation is unjustified, for the same patients are found

previously to have suffered, or subsequently to suffer, attacks un provoked by exercise or by other determinable cause And ques tioning usually reveals that the act of exercise originally blamed, even though it was particularly vigorous had been undertaken several or many times before without any adverse effect

It is reasonable to conclude that the burdens imposed by physio logical acts upon the normal heart, however heavy these burdens may be, never injure the heart's fibres, never produce injurious dilatation, and never exhaust the heart's reserve

Prolonged Overwork and Heart Failure

It has been thought that if the healthy heart is worked excess ively for prolonged periods, or if it is repeatedly called upon to do strenuous work, that it suffers, becomes diseased, and fails Heavy manual work has been regarded, with too little thought for its frequent association with exposure, alcoholism and infection, to be a main cause of cardiac failure, but acceptable evidence has never been a
heavy
straine
known The occasional racing horse or greyhound should not have been cited in the past Because a given hound won many races and ultimately developed heart disease and died, it is unreasonable to believe that the disease was the result of racing It is known that an increase in the amount of its work causes hypertrophy of muscle, whether somatic or cardiac Such hypertrophy does not constitute disease, it is physiological Our evidence points to there being no more than a slight physiological hypertrophy in athletes, there is no real ground for the belief that the heart becomes diseased in its hypertrophy, even in professional Marathon runners If racing dogs or race horses or racing men showed a much heavier mortality from heart disease than those that did not race, but were under conditions otherwise similar, this evidence would be suggestive Actually, it has not been shown that there is an increased mortality from heart disease in any one of these instances In the case of boat racing it has been proved that the expectation of life is unchanged, and that the subsequent mortality from heart disease is no greater than among the rest of the population So it will be wise to assume that the development of disease in the isolated instance is the result of a special circumstance and not of overwork To cite the isolated

case, without attempting to explain the exemption of the great majority, is to depart from the simple rules relating to cause and effect, a cause, to be acceptable as such, must always produce its effect when it acts in given circumstances

Coarctation of the aorta (page 268) contradicts the idea that failure easily follows from prolonged overwork In this congenital condition of obliteration or of great stenosis of the aortic isthmus, the systolic pressure may range about 200 or 250 mm Hg under conditions of bodily rest Here it might be expected that the early appearance of symptoms and signs of breakdown would be the rule This is not the case, for such subjects are known to maintain these pressures for very many years and to lead active lives, in which frequently these pressures are further and considerably increased, as also is the pulse rate If the heart can beat against high pressure in this disease for forty or more years without failing, and this seems often to be the case, then, even if failure sometimes comes, we should be rash to assume that excessive work is the simple cause We may recognise that the process underlying fatigue is cumulative, but there is no physiological sanction for an accumulation requiring years, let alone decades, to arrive at a critical level Since the heart fails to do to day the work that it has easily done for years, it is necessary to assume that an irreversible or permanent change has happened meanwhile in the muscle, and that the relatively rapid development of failure, if and when it comes, is the result of a change in the muscle that occurs, or occurs largely, in the latest period and not necessarily, or even probably, as a consequence of coarctation This change may be a change usual with advancing years, and one that accounts for the similar long delayed failure in essential hypertension, or it may be associated with intercurrent infections, we do not as yet know with any degree of finality The example remains a conspicuous instance of the uninterrupted overwork with which a muscle, presumably healthy, can cope effectively for very long periods of time

The case of the burden imposed by a valve defect has been dealt with briefly already (page 154), and it has been stated that patients suffering from such defects often enjoy long and active lives Such an example as aortic regurgitation may be used conveniently to illus trate the relation between work and heart failure, according to a conception that is of proved and great practical value I use the term "work", here as previously, in its ordinary meaning and not in the precise sense of effective "work done" In Fig 32 the full or

normal capacity of the heart for work is represented by the height of column A, which is divided into two parts, to represent the work required of the heart to meet the needs of the body under resting conditions (marked "rest") and the reserve The reserve is deliberately shown to be many times that required under conditions of rest In the second column (B), the heart is supposed to be meeting the needs of vigorous bodily exercise The work of the heart in this exercise is many times greater than that required of it during rest, a reserve is still shown though it is *greatly decreased* A hypothetical example of aortic regurgitation, the muscle of the heart being perfectly healthy, is represented in column C, and the column is divided into rest factor, aortic factor, and reserve The aortic is arbitrarily represented as equal to the rest factor, the work required of the heart when the body is at rest being no more than doubled, in such a case the reserve is little diminished, and the heart may be represented as still just capable of meeting everything that is required of it in vigorous *exercise (column D)* Column D represents the very rare case of aortic regurgitation, possessed of almost perfect exercise tolerance In columns E and F the capacity of the heart for work is represented as reduced, while there is an abundant reserve under resting conditions, this reserve is inadequate to meet the requirements of vigorous exercise, and exercise tolerance is diminished The capacity of the heart for work is represented as still more deficient in column G, the rest and aortic factors covering a considerable proportion of the whole column, and the reserve being greatly diminished The case is one in which only small calls for extra work can be answered and exercise tolerance is much reduced Lastly, in column H, the capacity of the heart is represented as so much reduced that this organ

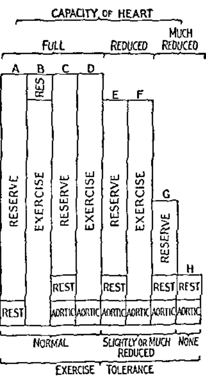


Fig 32

is incapable of meeting more than the requirements of rest and the aortic lesion The reserve has gone, the patient is breathless at rest, failure with congestion is beginning A main object of this diagram is to emphasise the idea that failure of the heart is not to be regarded as the simple result of increased work, but chiefly of decreased capacity to work The difference between the cases represented in columns C and H is not a difference in the aortic burden but a difference in the reserves of the heart

When heart failure develops it does so, certainly in the great majority of instances, because the capacity of the heart for work has decreased The extra burden of a valve defect is insufficient to embarrass the heart, it is merely sufficient to reduce the reserves The burden is not in itself lethal even when long continued But it hastens the onset of failure when the muscle is becoming weaker Heart failure often occurs, as previously stated, in cases in which there has been no evidence of an unusual burden The comparative immunity of the heart from failure in coarctation of the aorta in which resistance to the output of the left ventricle is continuously raised for many years, the similar immunity in cases of pulmonary stenosis, which throws a heavier load on the right ventricle than does mitral stenosis, are striking examples, the immunity is due to the burden arising out of a congenital and not out of an inflammatory or degenerative cause—the hypertrophied muscle is healthy and its strength therefore unimpaired

The manner in which failure is related to the work of the heart is not a mere matter of interesting discussion There is no branch of medical practice that requires more than that which deals with heart disease a simple and clear philosophy to guide it No doubt now remains that the treatment of the cases usually called heart strain and considered in the next chapter, has been gravely mishandled in the past, and that a large number of people who have been thought to suffer from this condition have been reduced quite unnecessarily, to a state of partial invalidism It is to the older philosophy which so greatly over emphasised the factor of strain or of mechanical defect as a cause of heart failure, that that unfortunate result is to be ascribed

CHAPTER XVIII

EFFORT SYNDROME BREATHLESSNESS OF EFFORT

Effort Syndrome (Athlete's Heart, Soldier's Heart)

When a healthy man is taking exercise that is sufficiently strenuous he experiences certain symptoms, and certain physical signs develop The most constant symptom is breathlessness, which comes during exercise, and continues for a variable time afterwards in gradually lessening degree He may become aware of his heart beat or of giddiness, he may actually faint After long or very strenuous efforts, the tremulousness and weakness of exhaustion are felt and, later, the lassitude and aches of fatigue Heart rate and blood pressure are raised during the period of exercise, and the rate and depth of breathing are increased, accessory muscles of respiration being brought into action The cardiac impulse becomes more forcible and is often diffuse, systolic murmurs often occur over the basal, and less frequently over the apical, regions of the heart The term 'physiological syndrome of effort' is applied to this group of symptoms and signs

The term "effort syndrome' is used to cover a similar series of symptoms and signs that occur in many patients complaining of ill health From the standpoint of symptomatology the difference between these patients and healthy people consists mainly in the ease with which the symptoms are brought on by exercise, and less frequently by emotion, though in the patient additional symptoms may appear The dividing line is not abrupt Very probably several distinct syndromes are being confused, certainly the syndrome is preceded by different primary conditions

The syndrome is essentially a malady of young people, but it is not confined to youth or to one sex Occurring in budding athletes or in young soldiers, it has attracted particular attention, because it renders these quite unfit to follow their ordinary occupations It is most important to realise that the syndrome is not peculiarly, or even particularly, a soldier's malady or an athlete's

malady, it is one of the commonest chronic affections of sedentary town dwellers It tends, however, to remain concealed in the latter and to be much less a matter of complaint than in active people When the young men of a nation are all called upon for military service, then a considerable proportion of those drawn from sedentary occupations are found to be temporarily or permanently un fitted for active work, and the commonest form of unfitness is this malady, which is revealed by the unaccustomed physical drill and mental strain to which recruits are soon subjected

Because of the essential change, namely, the augmented heartbeat it is a syndrome that is more commonly mistaken for cardiac disease than any other Among those who display it there is the very definite group of cases suffering from chronic infections, obvious or concealed, the chief being pulmonary tuberculosis, local pus infections of nose, ear, throat, or other part, infections of bowel or bladder A group is that comprising patients who are convalescing from acute infectious diseases such as pneumonia, pleurisy, infec tions of the gastrointestinal tract, bronchitis tonsillitis, or influenza *Transient symptoms are the rule rather than the exception in the* first days of convalescence, but they may be prolonged for weeks, months, or even years In most cases however, no infection is found, and there is none in the recent history Another group is that of sufferers from anxiety neurosis as was found during the period of the 1914 war, and upon which as a causative factor emphasis has more recently been laid A few cases are suffering from hyperthyroidism

SYMPTOMS

The onset is usually gradual or so far back as to be forgotten It may be more abrupt, in which case it has often followed directly upon an infection When it has begun abruptly, it is stated that acts of work previously performed can no longer be undertaken without abnormal discomfort Very rarely, the malady is said to have come after an unusually strenuous or prolonged physical effort, the subject believing himself at the time to be in vigorous health Breathlessness brought on by exercise is an invariable complaint and is the chief symptom, palpitation is very common Both these symp toms may be provoked by very small amounts of exercise and may be severe in the more serious cases Dizziness accompanying change of posture or the end of an effort is very common, attacks of fainting of the vasovagal type (page 109) *occur in a number of the subjects* As in the response of the normal to strenuous or prolonged work,

tremulousness, weakness, lassitude, and fatigue are often added, even after minor efforts, precordial discomfort, in the form of aching pain, soreness, and tenderness of skin and muscle, is frequent but rarely severe, and is produced or aggravated by exertion

SIGNS

I define the syndrome, which I have named, by the exaggerated response of respiration and of the cardiovascular system to exercise, it is an error to apply the term to conditions in which these re sponses are absent At absolute rest in bed and during sleep the respiratory rate is normal, but a simple hopping test (page 4) deepens the excursion and raises the rate, according to the severity of the condition, to between 30 and 70 per minute In normal subjects of the same age the respiratory response to this test is scarcely noticeable The heart rate in sleep is normal, but it is almost always found raised in subjects that are simply resting, and if they are excited or restless or are up and about, increased pulse rate usually draws attention A simple hopping test raises the normal heart rate of young people to 90 or 100 per minute, and this subsides in a minute or less In the milder cases of effort syndrome the rates jump to 120 or 130, and in severer cases to 150 or even 180 per minute, and the subsequent return to the pre exercise rate takes much longer than normal The systolic blood pressure, while normal at rest, is in general higher than is expected in these subjects when up and about, and is raised unusually by exercise, and falls slowly again as does the pulse Both pulse and blood pressure react unusually to emotion also

The main cardiac impulse, which is often forcible and quick, lies in its accustomed place, but the impulse as a whole is diffuse, appear ing widely in the 3rd, 4th, and 5th and even in the 2nd and 6th spaces The cardiorespiratory murmur is very frequent, as are basal [illegible] appear also but

under mental excitement The signs are often so striking that it is difficult to persuade those who have no special experience of the cases, or experience of X-ray examination in them, that the hearts are undilated.

The subjects are usually thin, often undersized, or presenting long, narrow, or flat chests A number show signs of nervous instability as evidenced by unusual emotional responses and anxieties Coldness of the hands and feet, and free sweating of these and of the axillae, is common

SIGNIFICANCE OF THE SYNDROME

The malady that has been described is not an entity, it is better that the picture should have been painted without a central motive to fix attention for if the condition is regarded as a specific form of disease, the right attitude towards the patient is lost This should usually be one of deferred judgment Each case requires to be considered on its own merit, a number will at once find places under well defined diagnostic headings such as anxiety neurosis or incipient tuberculosis, some will by development enter their well-defined categories later while under close observation Most of the cases, however, will remain under the term "effort syndrome", a term that deliberately signifies a group of symptoms rather than a specific disease, and which, by refraining to imply the cause, helps to maintain an attitude of enquiry towards the case

The symptoms are essentially provoked by effort, and are provoked very easily in severe cases It is for this reason, and because *certain cardiovascular manifestations are prominent, that the syn*drome came to be regarded in young men and soldiers as a disorder of the heart, resulting primarily from the strain of strenuous athletics or the long continued efforts of active military service The idea of heart strain found support in the diffuse impulse and the occasional systolic murmur, both being regarded as indicating dilatation of the heart The diffuse impulse is unreliable, and the systolic murmurs in these cases are exocardial more often than not, both diffuse impulse and murmur result simply from overaction of the heart, the former being rendered conspicuous if, as is common, the chest is narrow or flat, and thinly covered

Another factor that has contributed to the idea that the malady originates in effort is the not infrequent history that breathlessness had first been experienced with unusual exercise It seems often to be overlooked that the effort has been made many times before without unusual symptoms If the respiratory reserve is declining, or has declined, the first symptom will naturally appear with the most vigorous effort that is customary, that is the occasion on which the reserve is put to the test, and is found to be deficient The chief part that effort plays is clear, it calls upon a waning reserve, bringing a previously concealed defect to light I have seen no case among civilians or soldiers in which I could regard the evidence as convincing that unusually prolonged or unusually strenuous physical work was the primary cause of breathlessness

During the early days of the 1914 war patients of this type were regarded, according to views then current, as primarily cardiac and were rested, or, when exercised, were exercised with much caution Experience soon proved any fears groundless, many thousands were ultimately given graduated exercise rising to the full point of toleration, and in many to strenuous exercise In not a single instance was there suspicion of adverse after effects of this treatment, on the contrary, the exercises came to be used widely as remedial measures

PROGNOSIS

The prognosis of cases of effort syndrome in which no underlying infection is found is good, though progress may be slow Within five years and living under ordinary home conditions, a third of these patients are quite fit or are improved Most of the remainder remain in an unchanged state The sickness and mortality rates among the group are normal for their surroundings, with the single notable exception of tuberculosis, which is a little more prevalent than it should be Heart disease does not appear more frequently than is usual The younger the patients the greater is the percentage of recovery within a given period

MANAGEMENT

Cases presenting the effort syndrome should be closely searched for foci of infection when first coming under observation and, later, from time to time Local infection will demand appropriate treatment, but in most cases none will be found The patients should be persuaded that they have no heart disease, and that the sensations they have attributed to the heart have in reality another source, or are due to temporary and trivial disturbance of the organ consequent upon general lack of tone and fitness Very many have been perturbed by a cardiac diagnosis and are anxious about the future Simple reassurance of the patient and his friends often brings about improvement To diagnose heart strain in these patients and, on this or other ground, to forbid exercise or to enjoin rest, is to cripple these patients and to bring many of them to a state of partial or complete invalidism The most successful method of treatment is by simple exercises in the open air, for this encourages a more vigorous habit of mind and body The exercise, whatever [illegible] by the patient's responses In the

r very simple

so forth, *the*

patient should not exercise or be exercised to the point of distress but to a point that quickens and deepens respiration appreciably There are few cases that will not benefit from two periods of exercise, each of half an hour or more, each day The longer the cases are in the open air the quicker, in general, they improve, and so the hours of work among students and those employed in sedentary occupations require to be curtailed, and often may cease with profit for a period of weeks or months Exercise is pressed as recovery permits Breathing exercises are useful in expanding the chest and increasing the vital capacity of the lungs especially in young subjects The patients should be well fed Smoking interferes with recovery and should be forbidden or kept to the strict limit of a small smoke after each chief meal It is important in these cases to enquire about worries and as far as possible to remove sources of irritation or anxiety Treatment by drugs, other than occasional tonics or laxatives when required has little value

Differentiation of Breathlessness

The symptom breathlessness on effort is common to heart failure and to the syndrome now described The recognition of early heart failure displayed solely as breathlessness, and its differentiation [illegible]

effort syndrome it is associated with conspicuous overaction of the heart For the most part the differentiation is a simple matter therefore But there are instances of real difficulty The effort syndrome is a very common one and there is little reason to doubt that it often occurs in patients who actually display cardiac disease This is suggested not only on *a priori* grounds but because breathlessness in some young cardiac cases is out of all proportion to the signs and shows no tendency to progress to congestion It is most important clearly to differentiate because in general the treatment of failure consists of rest and in the effort syndrome exercise is to be encouraged The contrast in treatment, however, is not so sharp as this for limited exercise is beneficial in cases of *heart disease in which breathlessness is slight and exercise should* not be allowed to distress patients who are regarded as free from structural disease In practice, therefore it means only that the heart case is allowed a good deal less exercise than the other The

duration, and the pericardium thus serves to guard the heart from injurious over distension, such as would occur, for example, in asphyxia But the pericardium yields readily to chronic distension, whether this is brought about by an enlarged heart or by effusion The heart lies between the bodies of the vertebra and the sternum, and there are no intervening structures that are compressible, distension of the pericardial sac occurs therefore first to left and right, but especially to left, and from the left it distends backwards pressing upon and causing collapse of the basal part of the left lung

SYMPTOMS

Pericarditis is usually quite painless Pain, when present, is probably due to involvement of extrapericardial tissues, such as the diaphragm It may be a continuous severe precordial ache, or a stabbing pain reaching the abdomen or the left shoulder, and felt especially on inspiration Breathlessness resulting from obstruction of venous return or from pressure on a respiratory organ by large effusions is sometimes to be counted a true symptom, with other occasional pressure symptoms, but it is hardly possible in most cases to separate such from associated symptoms of cardiac failure The remaining symptoms result from the causal infection or accompanying disease, and these vary greatly in kind and in severity

SIGNS

When pericarditis comes as an acute infection, the dominant signs may be those of intoxication *Suppurative pericarditis* gives high fever and pulse rate, breathlessness, often great restlessness, and sometimes delirium As a part of a subacute rheumatism the toxaemia is less intense, though fever is the rule, restlessness common, and increased pulse rate invariable

Cardiac impulse —In effusion the heart usually maintains its contact with the chest wall, but the heart's impulse often changes nevertheless It becomes diffuse and wavy It may be displaced upward to the 4th space or outward Occasionally it disappears, and the heart sounds become more distant Such changes are not always the simple result of pericarditis, there may be simultaneous dilatation of the heart In children the precordial intercostal spaces may flatten

Distension of veins —The heart is very sensitive to intrapericardial pressure, which, like intrapleural pressure, is normally below atmospheric pressure A small rise of pressure in the pericardial sac at once interferes with the filling of the heart and causes a fall of

arterial and a rise of venous pressure Any considerable increase in intrapericardial tension greatly impedes the venous return and the veins become distended Distension of the veins is frequent in pericarditis with effusion but can rarely be shown to arise in the manner described, that is so because pericarditis is so often accompanied by changes in the heart that lead to failure with congestion

Cardiac dulness—In considerable effusion cardiac dulness extends up into the 2nd and then into the 1st interspace This new area of dulness may spread as much as 2 inches to the left of the sternal margin, and to the right may overlap the sternum It is the most important sign of fluid that we possess, it is best displayed by patients in the recumbent posture, and may lessen or even disappear in the erect posture

Cardiac dulness also increases laterally, especially at the level of the 4th and 5th spaces and both to left and right, the latter is the more significant To the left, dulness may appear well beyond the outer margin of the impulse The contour of the heart s dulness in large effusion is that of a blunt cone, right and left margin running obliquely but unusually straight, or it is pear shaped

Friction sounds are produced by the rubbing together of visceral and parietal membranes that have lost their polish or are actually rough The sound often simulates a blowing murmur but is usually shorter, seems more superficial, and ends more abruptly than the latter, usually, too, the sound has a characteristic scraping quality There may be a single systolic sound, and this usually begins a little later than systole, but it is the rule to hear a diastolic element as well to and fro sounds of equal intensity and duration The to and fro sounds do not change in relative intensity from place to place as do the conducted to and fro murmurs of aortic disease Very occasionally a third friction sound is added to make a sort of gallop rhythm of friction Creaking or leathery sounds are unusual Pericardial friction limits itself strictly to the precordium, may occur over the whole of it, but is more often confined to the central parts and base, it may be quite local It is seldom palpable

The friction rub is the only sign we have of dry pericarditis It may also result from chronic roughening of the pericardium, and so the advent of friction sounds or change in their character or extent from day to day, is more important, in fact alone conclusive of active mischief Although friction sounds often change in character, or disappear as fluid collects, the presence of a rub does not exclude effusion, or even large effusion, for the heart is often maintained in

contact with the anterior parietal pericardium in these circum stances

Friction sounds having a cardiac rhythm may also be heard when there is inflammation of the pleura overlapping the heart, such friction has a respiratory rhythm as well and is called 'pleuro pericardial friction" It is heard along the right or left border of cardiac dulness

Basal signs —In large effusions the lung becomes compressed at the left base An area of dulness appears, extending from the spine as far as the mid scapular line or a little beyond it, and upwards as far as the angle of the scapula This rather rectangular form is characteristic, as are the signs which accompany it The breath sounds at the base are weak or absent but just below the angle of the scapula loud tubular breath sounds and aegophony or broncho phony are heard, often accompanied by crepitations When these signs are early they will sometimes disappear if the patient leans far forward In recurrent effusion the signs may vanish and return several times In rheumatic children collapse may also accompany great enlargement of the heart without effusion

X ray signs —The shadow of the heart is widened laterally, the basal part of the shadow is increased in breadth and density, and its upper left margin is much more oblique than normal in the re cumbent position these signs become less conspicuous in the upright position Pulsation of the margin of the silhouette is reduced

Diagnosis of effusion —To differentiate between acute dilatation and pericardial effusion in rheumatic children is often impossible, especially since the two conditions often coexist The most import- ant guide to effusion is upward extension of dulness In adults small pericardial effusions are rarely diagnosed When large, the characteristic area of dulness and the signs at the base are usually sufficiently distinctive Friction sounds or the loss of cardiac im- pulse may draw attention to, and X ray examination will help to identify, effusion The extension of cardiac dulness well beyond the impulse is a valuable sign in favour of effusion When dulness to the right of the sternum is extensive this may result from a greatly enlarged left auricle (page 123), in that case heart sounds will generally be loud over it

COURSE AND TREATMENT

Acute suppurative pericarditis, whether it occurs as a primary or secondary manifestation of infection, usually runs a rapid and fatal

course Subacute serofibrinous pericarditis is usually rheumatic and heals spontaneously in most instances, but the course of the illness depends on the malady as a whole rather than upon that of the pericarditis Chronic forms of pericarditis with effusion run variable courses, but are subject to relief, and are from time to time cured, by tapping or by surgical intervention

Pain in pericarditis is best treated by local applications such as the ice bag, leeches, hot packs, or other mild irritants, and, if severe, by morphia Some of these patients find ease by kneeling, leaning forward upon pillows, which then support the chest

Paracentesis —Instances of undoubted pericardial effusion should be tapped if pus is suspected, and if pus is found the pericardium may be opened and drained surgically Simple aspiration may suf fice to give temporary or permanent relief in cases of chronic sero fibrinous or haemorrhagic effusion, and those that reaccumulate may be treated surgically In exploring the pericardium, a trocar and cannula is driven into the 5th space within the left margin of dulness in the direction of the centre of the chest for about 3 cm or a little more, or until the movement of the heart is felt In that case the trocar should be withdrawn at once, care being taken to allow no air to enter

Adherent Pericardium (Rheumatic)

Pathology

Adherent pericardium usually results from, and is common after, the rheumatic carditis of childhood or adolescence Pericarditis in healing leads to simple thickening of the pericardium locally, to local adherence of visceral and parietal layers or to complete union with obliteration of the sac The adhesions are for a time soft and friable, so that the two layers can be stripped apart ultimately they become tougher and less separable, but the membranes are not greatly thickened Simple obliteration of the sac is the commonest variety of chronic pericarditis

Similar adhesions may occur between parietal pericardium and the front of the chest wall, and may be accompanied by partial obliteration of the margins of the pleural sacs

Symptoms and Signs

The smooth glistening pericardial membrane lubricated by fluid normally allows the heart to move within the sac with little friction

Yet this smooth sac is not indispensable, as is shown by its occasional congenital absence, out of which no trouble seems to come Obliteration of the sac after rheumatic infection does not increase the work or weight of the heart appreciably It gives rise to no symptoms The intrapericardial adhesions may be responsible occasionally for a friction rub or cardiac crepitus, but they cannot be diagnosed, unless on the basis of a remembered acute inflammation of the sac

Extrapericardial adhesions of rheumatic origin may give rise to cardiac or pleuropericardial crepitus or friction, and to other signs

Systolic retractions —During systole the heart becomes smaller and tends to create a vacuum in its vicinity In front this pull is exerted, especially by the right ventricle, in the region of the 4th and 5th left rib spaces and in the epigastrium A heart of normal size, beating quietly and covered naturally by lung, pulls sufficiently to cause a little recession in the epigastrium only, and that not always Adhesions are credited with the production of systolic retractions of the ribs as well as of rib spaces The 5th and 6th ribs are chiefly affected and, through the diaphragm, the xiphisternum and lowest ribs along the left margin of the chest Retraction of the more rigid parts of the chest wall will naturally happen more readily when these are young and pliant, and will be the greater the more vigorous the movement of the ventricle, but there is no retraction that is not to be observed in the absence of adhesions There is no doubt that retractions are common in chronic heart disease following rheumatic pericarditis, but the subjects are for the most part young and the hearts much hypertrophied, and so it is probable that a vigorous movement of the enlarged organ contributes more than do the associated adhesions

Immobile impulse —Normally the impulse and left margin of cardiac dulness move 2 to 4 cm (1 to 1½ inches) to the left when a young subject rotates from the supine to the full left lateral posture, and equally to the right when the full right lateral posture is assumed Adherency of the heart to the chest wall naturally renders it immobile

Absolute cardiac dulness fixed —The absolute or, as it is sometimes called, superficial cardiac dulness maps out the portion of the heart that is uncovered by lung, it is bounded ordinarily by a line drawn down the left border of the sternum from the 4th rib, and by another from the 4th rib to the cardiac impulse This area is smaller in emphysema, and it is larger when the lungs have shrunk away

from the heart, and when the heart is enlarged Normally it disappears in full inspiration When the heart is bound to the chest wall by adhesions, the lungs cannot move forward to meet in front of the heart Such adhesions can be excluded if it is possible for the patient to abolish the absolute dulness by expanding the lungs But failure of the lungs to encroach materially upon the superficial dulness occurs with simple pleural adhesions The sign has most value when the left border of absolute dulness fails to move at all with forcible inspiration

SIGNIFICANCE AND TREATMENT

Simple adherency of the pericardium has probably no effect on the weight of the heart or upon the life of the subject The condition may be found in people who have lived long and very active lives, whose hearts are not enlarged and who have died of some quite separate malady As it is undiagnosable it has no clinical significance The prognosis, like the treatment, is governed purely by associated phenomena

When ribs are strongly retracted, the heart expends energy, some times very appreciable energy, in the process If the action of the heart remains embarrassed despite rest and other appropriate treat ment, removal of several precordial ribs (thoracostomy) has proved in some instances to be distinctly beneficial The operation is less practised than formerly

CONSTRICTIVE PERICARDITIS

PATHOLOGY

This is a comparatively rare chronic malady, evidently inflamma tory but usually insidious in origin and rarely attributable to a particular infection It is thought to be tuberculous occasionally, sometimes it follows an acute pericarditis A rheumatic history is quite unusual Disease of the valves is not found, but auricular fibrillation is sometimes associated It is found chiefly in adolescents and young adults of both sexes, though the ages of the cases have ranged from 5 to 50 years The pericardium is much thickened by very dense and thick fibrous tissue, laid down uniformly or in the form of one or more strong and wide bands The new substance, unyielding in its almost cartilaginous consistency and sometimes containing large calcareous deposits restricts the movements of the heart, embarrassing systole and impeding diastolic filling The

fibrous tissue often spreads beyond the pericardium The lateral parts of the pericardium, with the adjoining layers of pleura are welded together to form a firm partition between heart and lung, stretching from the root of the latter to the diaphragm The same process invades the roots of the lungs, the base of the heart and its outgoing and incoming vessels and the posterior mediastinum, further anchoring the heart and sometimes constricting the main vessels When so extensive it has been termed mediastinopericarditis The pericardial sac may be completely or partly obliterated

SYMPTOMS AND SIGNS

The symptoms of great thickening of the pericardium and of the extrapericardial adhesions which usually accompany it are mainly those of venous congestion including swelling of the abdomen The signs that may occur are many

Venous engorgement —Venous congestion and its accompaniments forms perhaps the most important manifestation in these cases It arises out of inability of the heart to expand adequately owing to its encasement by unyielding fibrous or calcareous tissue and at times out of actual constriction of the veins The congestion of the veins is conspicuous, cardiac pulsation in them is not free and the congestion is out of usual proportion to the degree of breathlessness and to the enlargement of the heart The presence of intensely engorged veins in youngish subjects in the absence of signs of valve disease, of much cardiac enlargement or of much breathlessness is of particular diagnostic importance Enlargement of the liver too is unusually prominent, due, so it has been thought, to direct interference with the hepatic veins Great enlargement, cirrhosis of liver, and extreme perihepatitis are sometimes displayed, and are possibly to be associated with long continuance of congestion possibly with perihepatic infection Ascites is so common and prominent that many cases are suspected to be the subjects of hepatic or peritoneal disease primarily Dropsy of the legs is less common, the face may become swollen

Inspiratory swelling of veins —The cervical veins normally collapse in inspiration because blood is sucked into the heart when thoracic pressure falls In some cases of mediastinopericarditis an obstruction to the entry of blood occurs during inspiration, presumably because fibrous bands tighten about the heart or about the great veins, thus it happens, paradoxically, that the veins swell in inspiration.

Inspiratory decline or failure of pulse —In mediastinopericarditis, inspiration of ordinary depth sometimes interferes with the filling of the heart, as has been seen Such interference will cause the pulse to become smaller and pressure in the arteries to fall In the same circumstances a deeper inspiration may sometimes obliterate the pulse It is important to note that all the pulses in the body are affected in the same way Because the heart beat remains strong and the pulse is weakened, the phenomenon has been called the "pulsus paradoxus" It is not a good name Its clinical value is much lessened because the significant point is not the decline in pulse strength in inspiration but the manner in which this is brought about, and the manner is not always obvious When it was believed that blood pressure rises in inspiration, a falling pulse in inspiration seemed to contrast sharply But this physiological teaching has proved erroneous The pulse beats fall away in many normal people and in many abnormal states when inspiration is deep and especially when it is abrupt This falling away is due to blood being caught up in the chest, the vascular capacity of heart and lungs being much increased when the intrathoracic pressure falls

In healthy people, too a deep inspiratory movement not infrequently causes the subclavian artery to become nipped between 1st rib and clavicle, thus reducing or abolishing the radial pulse It is to be noted that the pulse declines or fails only in the arm in this instance

It will be evident that the response of the pulse to respiration in normal and abnormal cases is complex, and the sign discussed is only of value if closely studied and fully understood

Systolic retractions —In constrictive pericarditis movement of the chest wall with the heart beat is often slight, in other instances systolic recessions may be as emphatic as those displayed by the rheumatic heart

It is not quite correct to regard the heart as tugging the precordial ribs inwards through firm adhesions between it and the chest wall When the heart tends to draw away from the chest wall, the latter must inevitably follow it whether bound by fibrous tissue or not, provided that the lungs cannot move in to fill the gap [illegible]

pericardium and pleura separates the heart from the lungs, the

heart expends energy in pulling in all directions upon relatively unyielding structures Thus, fibrous tissue covering the heart laterally impedes its beat more than do any adhesions anteriorly, for the former alone guards a yielding structure

Immobile impulse and absolute cardiac dulness —The enclosure of the heart in firm partitions of fibrous tissue render immobile such impulses as are displayed and adhesions securing the heart to the front of the chest wall will prevent as already explained change in the absolute cardiac dulness during respiration

X-ray signs —In great fibrous thickening of the pericardium the borders of the heart are straighter than normal and the points of junction of vessels and different chambers tend to be indistinct The rounded apex of the heart is not seen free of the diaphragm Irregular shadows may be seen projecting from the silhouette of the heart or basal vessels interfering with its normal outline and with the sharpness of its margins The diaphragmatic movements are often greatly limited, especially in the region of the heart's apex, which moves little or not at all Sometimes definite distortions of the diaphragm are observed, especially directly to the right of the heart, during inspiration, where adhesions may sometimes be seen Pulsation of the silhouette, especially of the right margin, is reduced or abolished Immobility of the heart with change of posture can be identified A well exposed film may exhibit a calcified sheath upon the heart

Electrocardiogram —The curves are nearly always of low voltage, or present flat or invert T waves

DIAGNOSIS

The diagnosis of constrictive pericarditis depends chiefly upon observing intensely engorged veins in young subjects in the absence of signs of valve disease, of much cardiac enlargement, or of much breathlessness The possibility should always be considered in cases of enlargement of the liver or of ascites that are unexplained

PROGNOSIS

Constrictive pericarditis is essentially a chronic malady Life may be continued for many years in a state of partial and unchanging invalidism, but usually complete invalidism is the result after a few years and the outlook is then grave

SPECIAL TREATMENT

In cases in which the heart expends much of its energy in pulling upon resistant structures and exhibits this by powerful retractions of the chest wall, relief may be obtained by simple thoracostomy, removal of the affected ribs and cartilages When the heart is found to be encased by dense tissue or to be held by constrictive bands, surgical treatment should be more drastic The operation consists in removing the 4th, 5th, and 6th ribs and cartilages and part of the sternum over the precordium and in resecting such parts of the thickened pericardium as can be taken away safely from the front of the heart and from its diaphragmatic surface Sometimes a line of cleavage can be found and the thick covering layer removed by blunt dissection in other instances it must be cut away with the utmost care In skilful hands the mortality has not been high, and the operation has met with conspicuous success in a considerable proportion of cases, all symptoms and all signs of venous congestion disappearing and the patients resuming normal activities In other cases improvement has been definite but cure has not resulted

CHAPTER XX

BACTERIAL ENDOCARDITIS

SUBACUTE BACTERIAL ENDOCARDITIS

THE subacute *variety of bacterial endocarditis is* here emphasised because it is much the commonest form The malady is most prevalent between the ages of twenty and forty and is commoner among males than females

PATHOLOGY

Subacute bacterial endocarditis is due to an infection of relatively low virulence, which attacks valves that are already abnormal The bacterium is almost always the non haemolytic streptococcus viridans an organism that commonly inhabits the throat and is known frequently to invade the blood stream in subjects having many decayed teeth This organism is not virulent, it grows poorly in culture The vegetations are almost confined to the left side of the heart, the side on which chronic valvular disease is common The chief exceptions to this rule are cases of congenital malformation in which the right heart is involved as in patency of the interventricular septum, pulmonary stenosis or patent ductus arteriosus The disease ordinarily attacks the mitral or aortic valve In most cases one or other of these, and usually the aortic valve, is the main seat of the disease, both may be heavily affected and, if the valves are searched for minute vegetations, it will be realised that the cases are few in which either valve escapes completely In a considerable proportion of the cases of aortic valve involvement, these valves are congenitally bicuspid, this deformity occurs in little less than 1 per cent of all autopsies, and about a fifth of all such cases of congenital malformation acquire infective endocarditis, it is therefore a notable factor in determining the disease In other cases it is clear that vegetations have formed on old standing rheumatic disease of mitral

or aortic valves, a history of rheumatic fever or chorea is present in a high percentage of the patients Syphilitic disease of the aortic base is also found, though less frequently

Clinical observations show that cardiac patients who acquire this disease do so for the most part while in good or fairly good general health, cases of congestive failure are relatively immune It is the rule to find numerous vegetations and not a single mass, the vegetations often involve all the cusps of either aortic or mitral valve or both The vegetations are mostly small, warty, or shortly pedunculated, some are much larger, club shaped, strap like, or form large irregular masses, they affect the lines of apposition of the cusps first of all, but in heavier involvement plaster the whole surface of the cusp and appear in smaller or larger confluent masses upon the walls of auricle or ventricle, upon the lowest parts of the aortic wall, and upon the chordae tendineae, some of which are frequently cut through The parts of affected cusps that are free of actual vegetations are nearly always thickened, often much fibrosed and deformed, and sometimes show healed perforations Often vegetations are found that are in an advanced stage of fibrosis, strap like vegetations are always tough at their bases, calcareous deposits are not infrequent In such cases, and they are those that have run long courses, the appearances indicate that a process of new infection by contact or spread, and a process of repair, continue to challenge each other in a fight for predominance, which is carried on over long periods of time Sometimes freshly broken vegetations are found where emboli have become detached

CHIEF CLINICAL MANIFESTATIONS

Onset —The malady, regarded as an infection generally begins in early adult life, and its onset is usually insidious, so insidious that sometimes it may be many months before the subject realises his health to be seriously affected The usual early symptoms are lassitude and weakness, combined perhaps with occasional feelings of chilliness and malaise, perhaps with aches about the joints or muscles, these symptoms continue and the patient passes gradually to a state of debility and anaemia Anorexia with or without nausea, and later vomiting, is an early symptom in some cases The first symptoms may be those of an acute febrile illness with chills and sweating The first symptoms may arise from embolism of one of a number of important vessels, giving a varied and sometimes puzzling symptomatology.

The phenomena of the established condition are conveniently described in three groups Firstly, there are the local signs in the heart corresponding to old standing mischief and to new damage of the valves consequent upon their recent inflammation Secondly, the *disease may be regarded as equivalent to a* condition of chronic septicaemia or intoxication most of the manifestations are properly placed in this category Thirdly, there are the very varied effects of embolism

Local signs —Since the disease is one that attacks valves often much deformed already, and as the disease itself damages the valves further, perfectly clear signs of valvular mischief are almost always to be obtained The cases are generally those of aortic regurgitation or of mitral stenosis the remainder are interpretable as mitral regurgitation or are instances of congenital heart disease When there are no signs pointing emphatically to disease of the valves, signs of definite or moderate enlargement of the heart will usually be found It will be understood that the degree in which these local signs are attributable to the disease we are considering can be estimated clinically only if the state of the heart before its infection is *precisely* known

That the valves are actually the seat of active mischief is sometimes betrayed during the course of the malady by signs of new local damage, as when a murmur indicative of aortic regurgitation or of mitral stenosis changes its character conspicuously, or actually develops These changes, however, are witnessed very infrequently and therefore have little clinical importance

Symptoms and signs of infection are numerous and most of them are common to this and similar infections They include weakness, gradual loss of flesh, and many manifestations that require special mention

Fever is the rule, though it is often inconspicuous In the early stages of the disease, and particularly when the onset is insidious or the course long drawn, the temperature may be raised only 1° to 3° F, and there may be periods of days or weeks during which it is normal It is rather the exception than the rule for temperature to be raised in isolated readings taken in the daytime from patients who are up and about The temperature chart of bed cases *is very variable*, almost any type of curve may be shown but low grades of fever are the rule, high remittent or high continuous fever is rare before the terminal phases of the malady are reached

Pallor is not invariable even when infection has long established itself A few patients have the full and fresh colour of rude health But pallor is the rule and is often conspicuous The tint is variable, as is that of the bloodless normal skin, a special reason for variation is found in chronic cases, which acquire a distinct but diffuse cutaneous pigmentation A yellow pallor is usual in blondes, pale brown or muddy tints in those pigmented spontaneously or by the disease Pallor is due largely to lowness of red blood cell count, which is commonly near to three million or less, this is often aggravated by a hypochromic condition as shown by a low colour index

Leucocytosis is the rule, the count of white cells being usually between 10,000 and 15,000, occasionally much higher, occasionally normal or actually lower than normal The differential counts vary, but polymorphonuclear cell increase is much commoner than lymphocytosis

Splenic enlargement is present in almost all cases, and the spleen being firm is palpable in most cases, this is a very important sign The tip of the spleen is just palpable or comes three or four fingers' breadth below the costal margin, exceptionally it is much bigger and fill [illegible] ement

[illegible] ability to long lasting toxaemia, as it is in phthisis and bronchiectasis Swelling is confined to the last phalanges of the fingers, the swelling may be anteroposterior and accompanied by increased convexity of the nails or it may be lateral It does not produce really bulbous fingers, such as appear in congenital heart disease, a noteworthy contrast is that these clubbed fingers in subacute endocarditis rarely exhibit cyanosis Clubbing of the toes occurs, but is more difficult to recognise

Petechiae, scattered in small numbers, and appearing often in crops, are usual, they are especially frequent above the clavicles and around the axillae, on the upper arms, chest, and abdomen They are scarlet red brown, or brown, according to their age, and they last a few days They are to be distinguished from the small bright crimson angiomata that are so common in the skin, and which are either distinctly elevated or, if smaller, blanch a little or much on pressure When petechiae are seen in the conjunctival or buccal mucous membrane or under the nail, where they are often linear, there can be no confusion with the bites of parasites Petechiae should not be regarded as embolic, the minute vessels of the

skin in this state permit an escape of blood more easily than normal, the petechiae can be produced by congesting the arm Though occurring so often in endocarditis their diagnostic importance is much decreased by their frequence in many other conditions.

Purpura occurs occasionally and may be widespread, but is usually found as a symmetrical eruption on the legs, it occurs in the later stages of the malady, as do rare haemorrhages from mucous membranes Other cutaneous eruptions, including urticaria and different forms of erythema, are occasional

Joint pains and tenderness are common as transient disturbances, effusions are infrequent, but may affect single joints or many, the chief joints of the lower limbs are most often affected Pericarditis and pleurisy occur rarely

Renal involvement is common Abnormalities of the urine, such as the presence of a trace of albumen, hyaline casts, or pus cells in small numbers, are frequent Occurring alone these changes are insufficient evidence of nephritis Red blood cells are usually present, their appearance in small numbers is probably due to minute haemorrhages in the kidney, and occasionally to embolism A degeneration curiously limited to a part of the glomerulus is frequent and sometimes affects these structures in large numbers In the later stages clear evidences of parenchymatous nephritis are not infrequent, the urine is loaded with albumen, renal oedema occurs, and uraemic manifestations may appear

Cardiac failure with congestion is rarely present in the early stages of this disease, which attacks those who are in relatively good health, but it is so frequent in the later stages as to form a chief cause of death, it is always terminal Failure with congestion in these cases is the end result neither of the original burden imposed upon the heart nor of its increase through an aggravation of the valve defects The total valve defect is often slight, and the failure develops while the patient is at rest in bed, and is an obvious accompaniment of infection The disease affords one of the clearest illustrations of failure being determined by an infective process (page 30), but as there are no recognisable lesions in the heart muscle that can be called to account for this failure, the weakening of the muscle is to be attributed to poisoning.

Embolic accidents —The emboli are derived from the heart, a large vegetation, or a considerable portion of it, becomes detached, or it breaks into a number of pieces, and vegetation or its fragments

are swept on with the blood current The vegetations being almost confined to the left heart, embolism is almost confined to the systemic system The size of the embolus determines the size of vessel that can be entered and plugged The direction of arterial branching may be disregarded, and the travelling embolus considered as merely part of the current of blood No artery is immune from embolism, and chance chiefly determines which is affected, but blocking of certain vessels has conspicuous effects, thus attracting unusual attention and giving a false idea of the relative frequence of embolism in these

The chief effect is interference with local blood supply in instances in which a proper collateral circulation fails to become quickly established Embolism of the main vessel of a limb may cause gangrene, of the middle cerebral artery, hemiplegia, of a coronary artery, sudden death, of a retinal artery, partial blindness Embolism of a splenic vessel causes infarction of that organ, and may cause local pain and tenderness, and audible friction in the splenic region Infarction of the kidney brings albumen and blood into the urine, pain into the back, and sickness, and, rarely, when blood is abundant and clots form, causes symptoms resembling renal colic

The emboli of infective endocarditis often give rise to symptoms and signs unseen in the case of uninfected clots The events are well illustrated in superficial vessels An artery like the radial or dorsalis pedis is blocked by an embolus This may happen so unobtrusively as to escape any attention It may be noticed on careful search that [illegible]

tissues around the artery for a longer time In a few cases chronic arterial and periarterial inflammation continues, the vessel wall gives way, and an aneurysm forms locally Abscesses very rarely occur These different disturbances illustrate the varying power of the tissues to deal with the infection Essentially similar changes occur also in deeply seated vessels for example, aneurysms form on branches of arteries supplying the viscera

Infected emboli in smaller arteries, like the nasal, may give conspicuous inflammatory redness and swelling locally Digital nodes are small red or purple spots a centimetre or less in diameter, always tender when fresh, sometimes raised and often just palpable, and occurring in the pads of fingers and toes They last a few days and are important diagnostically Similar but more extensive lesions also

occur in the skin of the thenar and hypothenar eminences These cutaneous or subcutaneous lesions are thought to arise from infective emboli of small arteries or arterioles Similar emboli may probably account for occasional pain and tenderness appearing in muscles and joints

DIAGNOSIS

The diagnosis of subacute bacterial endocarditis has been obscured in the past by failure to recognise the insidious nature of the complaint and by the expectation of finding high temperature or at least some fever Misinterpretation of three of the cardinal

these signs are clubbed
lults presenting definite
of the fingers has been

regarded wrongly as a simple association of this heart disease In about 90 per cent of these cases clubbing of fingers is due to infective endocarditis though not diagnostic this constitutes one of our most valuable signs of the disease Occasionally it is the result of congenital heart disease the fingers then being cyanosed and their tips more bulbous very occasionally the clubbing is itself congenital and the patient will then usually be sure that the fingers have always been misshapen swelling at the base of the nail, due to irritation from breaks in the skin of the lunula should not be confused with clubbing An enlarged spleen has long been regarded as a consequence of cardiac failure Now the spleen unlike the liver, has a muscular framework and it is unusual for it to increase much, if at all, in size in congestive failure In chronic heart disease an enlarged spleen generally means infective endocarditis though its significance is less in countries where malaria is prevalent A palpable spleen is not unusual in uninfected cases of mitral stenosis Pallor has been looked upon as a common result of aortic regurgitation Aortic reflux does not produce pallor when the two are found together, an adequate cause for pallor must be discovered when pallor is conspicuous it is usually due either to infective endocarditis or to rheumatic infection, according to whether the patient is an adult or a child

In identifying infective endocarditis, the first requisite is clear evidence of cardiac disease Without this the diagnosis will contain an element of doubt that can scarcely be resolved before signs of embolism occur and streptococci are found in the blood

Given this evidence of heart disease, the presence of continued or periodic fever always arouses grave suspicion of the disease The addition of splenic enlargement increases the suspicion, but a diagnosis cannot yet be made, when heart disease is complicated by

sometimes be taken into account, the presence of clubbed fingers, signs of embolism, or a positive blood culture will settle the diagnosis in each instance Irrespective of other signs, if the patient is afebrile and remains so for many weeks, the diagnosis becomes highly improbable and is shortly ruled out

the diagnosis almost certain, even when at a single examination the temperature is unraised

In a heart case embolism combined with fever, splenic enlargement, or positive blood culture suffices, or it is enough if the emboli are known to be infected, or if digital nodes occur

A blood culture showing nonhaemolytic streptococci is insufficient for a diagnosis, even when heart disease is present, but if fever,

COURSE AND PROGNOSIS

The course of a disease presenting such a variety of symptoms

recovery have not always been substantiated, but a few are well authenticated The course from onset of symptoms till death averages six months, the termination may come earlier, or it may be delayed not unusually for eighteen months and occasionally for two, and rarely three, years Inconspicuous fever and frequent and long apyrexial intervals are usual in the cases running long course

Death occurs from progressive weakness and exhaustion or from cardiac failure in about equal numbers of the cases, and these together form about two thirds of the whole Serious embolic accidents, nephritis, and intercurrent maladies, including pneumonia, account for most of the remainder

TREATMENT

The patients should be in bed when febrile or while under active treatment, they can move about when afebrile, strolling quietly or sitting or lying out of doors in sunlight and in warm weather, according to general fitness Their food should be sustaining, light as a rule, heavier when tolerated It is important for them to be occupied, the visits of cheerful but quiet friends are very helpful Symptoms are met as they arise

Very many antiseptic remedies have been tried, almost all have failed and none has as yet stood an adequate test of time Sulphapyridine (alternatively, sulphathiazole) has brought a new ray of hope Given in full doses it will usually bring the temperature down, and cases remaining free of signs and symptoms of infection for several weeks or months have been recorded The drug should be given to an adult in doses of 6 g daily for 1 or 2 weeks and continued in 4 g doses to the end of the month The white cell count must of course be closely watched, and the drug discontinued if the polymorphonuclear cells show an appreciable decline The drug is sometimes combined with intravenous heparin, under the hypothetical idea that the organisms will be more exposed if no fresh clot forms, this use of heparin is of doubtful value and it may lead to fatal haemorrhage Penicillin is well worth thorough trial, successes have been reported under daily intravenous doses of 100,000 to 200,000 units for 2 or 3 weeks In the event of success with either drug, foci of infection should be sought diligently and eliminated under sulphapyridine to decrease the chance of reinfection

Where infection has occurred in persistent ductus arteriosus the ductus should be ligated This surgical procedure succeeds increasingly.

Acute Bacterial Endocarditis

PATHOLOGY

This form of bacterial endocarditis can occur at any age It often originates in a serious local infection in some other part of the body,

as in pneumonia, osteomyelitis, gonorrhoea, or puerperal sepsis

The bacteria responsible are usually the pneumococcus, staphylococcus aureus, gonococcus, or haemolytic streptococcus, other organisms occur occasionally The valves of the right heart are much oftener affected than in the subacute infection, the aortic valve is attacked most frequently of any, and after this the mitral One valve is usually the seat of disease and often one cusp The valve attacked may be the seat of previous mischief, but usually this is not the case, and the uninfected cusps, or parts of the cusp, have their normal thickness and translucence The vegetations are soft, usually large, not infrequently forming a cauliflower like mass Spread to the septa is occasional only Ulceration is frequent and cuts deeply, often perforating the valve The tissue reaction is acutely inflammatory Signs of repair are few both in the vegetation and around its base, this is so because resistance is slight and the disease runs a short course

CHIEF CLINICAL MANIFESTATIONS

Onset.—Developing in pneumonia or as a complication of other grave infection the onset is inconspicuous In cases that are primarily cardiac the onset is usually with high fever and its accompaniments—headache, vomiting, malaise, chills, and sweating

Local signs.—Signs of valve disease may be present from the start, but more often develop, a systolic murmur appearing at the cardiac impulse, or to and fro murmurs at the base, the latter may be to right or left of sternum and due to involvement of aortic or pulmonary valve Frequently there are no signs of valve disease Change in the character of a murmur is noted occasionally Cardiac enlargement, unless existing previously, is not the rule Inconspicuousness or lack of cardiac signs is responsible for the chief difficulties of diagnosis

Symptoms and signs of infection.—These may be enumerated as for the subacute form, though much more briefly The contrasts between the two diseases are chiefly due to greater severity of infection and the shorter course run in the acute variety

Fever is constant, is continuously high, remittent or intermittent, temperatures of 103° or more (up to 105° or 106°) are reached almost daily Rigors are common and profuse sweats the rule The gravity of the malady is shown by the prostration of the patient, by his apathy, or by delirium or coma Pallor is usually inconspicuous profound anaemia is rare, clubbing of the fingers scarcely occurs,

time is too short The polymorphonuclear leucocytosis is considerable the white cell count being from 20,000 to 30,000 The spleen presents signs previously noted, but, being soft, is not so often felt as in the subacute disease Petechiae and purpura are common The joints are less often, and the pericardium and pleura more often involved than in the subacute disease Congestive failure is unusual Albumen and red blood cells are often found in the urine and acute, but not chronic, nephritis may develop

Embolic accidents —These are very frequent, and the detached fragments are mostly large By obstructing the blood flow through important arteries, they cause corresponding symptoms, already described in detail Because the valves in the right heart are often affected, pulmonary embolism is not unusual In superficial arteries an inflammatory reaction is the rule and embolic abscesses are common Digital nodes are not a feature of this disease

DIAGNOSIS

The disease is more difficult to recognise than is subacute bacterial endocarditis That is so, because signs in the heart are found with far less frequency, and the diagnosis then comes to depend almost entirely on evidence of embolism Without cardiac signs or emboli, the disease cannot be differentiated clinically from simple septicaemia In pneumonia, in which the original temperature curve is continued or rises, the diagnosis will waver between endocarditis, tuberculosis, and empyema Continuous temperature lasting for several weeks, accompanied by increasing weakness, delirium, and splenic enlargement, produces a picture like typhoid fever, though [illegible] *differenti* [illegible] be kept in [illegible] e found in central empyema, and perinephritic abscess, must be remembered Blood cultures are always positive, the organisms growing readily.

COURSE, PROGNOSIS, AND TREATMENT

In the severest infections and in cases in which the patient is already the subject of grave disease, death may occur within a few days In a young soldier, in whom the disease was implanted upon a bicuspid valve, death occurred within 36 hours of the man reporting himself sick after a route march The usual course is a *few*

weeks, sometimes two, and rarely three months Death is generally the result of poisoning the patient weakening progressively Embolism of brain or heart occasionally kills Recovery is almost unknown The patients are mostly helpless and require devoted nursing—to administer food in liquid form through lips often unresponsive, to relieve high fever by tepid sponging to keep clothing dry amidst drenching sweats, to maintain cleanliness, and yet not to hasten the exhaustion, which comes so quickly despite all care

CHAPTER XXI

RHEUMATIC CARDITIS

Introductory

To understand rheumatic heart disease, as it is seen in its developed forms, it is necessary to study its evolution The conception, still prevalent, that what we call by the name "rheumatic fever" is a disease of the joints, and that this disease is frequently complicated by involvement of the heart, cannot be justified The disease is a widespread affection of the body, usually, it is true, but by no means always, producing arthritic symptoms To regard the joints as the primary or central point of the disease is to go beyond the facts To convey the idea, by the use of the traditional and almost always unsafe word "complication", that something is added which is not essentially a part of the disease, is to assume more than we know, and probably more than is true

The modern conception of the disease, when regarded as a specific malady, is of an organism (bacterium or virus) that invades the *blood stream, damages the small arterioles of the body, and sets* up a condition of inflammation, acute or subacute, which calls attention to itself especially in and around the joints, in the heart as a whole, and in the subcutaneous tissues and tendons The clinical picture is very varied, but the most notable manifestations are the intoxication, arthritic and cardiac troubles, and a display of nodules beneath the skin To this brief list chorea should probably be added, though its place in the syndrome is not yet clear Although arthritis, carditis, nodule, and chorea may each be in chief evidence, or appear as the only obvious clinical accompaniment of the infection, any two of these may be paired, and often three are present simultaneously or in other instances within short times of each other in a given case

The disease is specially one of childhood, but age modifies it, chorea, nodules, and carditis becoming much less frequent shortly

before or at the time puberty is reached, while disturbances of the joints increase in severity and acuteness to become the dominant feature of the disease in the third decade, or early adult life The name rheumatic fever is apt to conjure up the picture of the acute and relatively transient illness of the adult, but, while the heart may and often does suffer damage in such attacks of the disease in the third decade of life, rheumatic heart disease begins almost always in childhood In childhood the disease strongly tends to be relapsing and therefore to run a chronic course The full active course is often run, and life often brought to an end, before the child comes of age Rheumatic heart disease as it is seen in the adult consists in very large part of the damage arising out of the inflammations of these early years Mitral stenosis is the result of such damage, this and other injuries of the kind are sometimes found in patients in whom there is no history of illness to account for them, but if chronic rheumatic heart disease is to be seen in the actual making, to be followed through the stages of its progress, it is to the growing child that we must go.

Rheumatism of Childhood

The disease is one of cold and damp countries of the northern hemisphere, and is found particularly among the overcrowded and poorer families of towns It appears especially in the winter months The age period begins at about five years and rises to a peak at ten, and subsequently and gradually declines It affects girls more often than boys

PATHOLOGICAL ANATOMY

In fatal cases the pericardium is nearly always affected in some degree Its surface is congested and dull, and there are small deposits of fibrin upon it Often the pericardium is universally inflamed and swollen, and covered by a thick layer of fibrinous material Pericardial fluid may be in excess and is turbid, but in children is rarely large in amount In cases that have run a long course, universal soft adhesions are common

Whenever the valves are involved, and this probably happens always in a case of carditis, the mitral and aortic valves are usually both affected in greater or less degree, the mitral valve is probably involved invariably and usually heavily, the aortic valve is involved a little less frequently, usually less heavily, but sometimes most

heavily The tricuspid and pulmonary valves come next in this order, the former being involved in as many as a third of the cases if we count slight disease and the latter infrequently

The lesions on the surface of the valve consist of the almost characteristic small sessile vegetations set in a band along the margins of apposition and involving the whole circumference of the valve when this is heavily invaded, and occasionally affecting other portions of the valves, such as the chordae tendineae Beneath the surface is the more notable spreading inflammation in the valve substance These lesions represent the active stage of the disease, as this attacks in the first instance healthy valves and in recurrent attacks valves already scarred by similar past injuries In healing they leave behind thickened, shrunken tissues narrowed orifices, incompetent valves as previously described in detail (pages 131 and 140)

In most cases cloudy swelling of the heart muscle is present In cases running a longer course this is less in evidence and the sub miliary nodule is the characteristic lesion These nodules present a fibrinous or necrotic centre and contain large bipolar or multi nuclear darkly staining cells young fibroblasts lymphocytes and a few leucocytes They are frequently found around small blood vessels and occur in the interstitial tissue of the auricles and ven tricles especially around the valve rings in the subendocardial tissue, in the pericardium, and in the aortic adventitia They may be few in number or the heart may be crowded with them They also occur in many other parts of the body, especially in the periarticular tissues They have been thought to be peculiar to rheumatic inflam mation but are not quite so In healing they leave behind at the most a minute fibrous scar The subcutaneous nodules, or granu lomata, though much larger, are composed of similar tissue elements, and evidently represent a similar form of subacute or chronic in flammatory reaction

SYMPTOMS AND SIGNS

Onset—Although the disease may begin abruptly with fever, an insidious onset is commoner The child is brought with the complaint that it feels poorly, has lost appetite, is easily tired is pale, and has been losing flesh, there may be pains in the limbs or chest

Pulse and fever—In this disease the pulse and sedimentation rate are more valuable guides to the presence of infection than is the

temperature chart Fever is often absent from both short and long phases of active mischief, but the pulse rate is always quickened In febrile cases too, the temperature is usually low and often phasic. It often rises as joints or heart become involved, it almost always rises in pericarditis, it falls away by lysis Temperature charts show much variation A phase of fever lasting ten to thirty days may be isolated, or may be repeated one or more times with intervening periods in which there is no fever, but during which the pulse rate is usually raised Cases in which short and slight phases of fever occur over a period of six or nine, or even twelve or eighteen months, are not uncommon Hyperpyrexia is rare In general, the longer the course the less fever is in evidence There may or may not be leucocytosis Pallor, often rapid in development, is usual and may or may not be associated with actual anaemia

Joints —Involvement of joints is the rule, often it is inconspicuous and manifests itself by pains only Usually there is a little swelling and tenderness of such joints as the wrist or knee When several joints are involved, they are often involved in succession

Carditis and its manifestations —Even in first attacks the heart is invaded in a high percentage of cases, it is probably always involved in prolonged or recurrent infections of low grade In cardiac involvement a rise of pulse rate is conspicuous and customary, and there is breathlessness The cardiac dulness enlarges laterally, the impulse moves out, the 1st sound becomes less distinct, a systolic murmur is heard in the apical region The diastolic murmur of aortic regurgitation may appear to the left of the sternum Friction lasting a few days is not uncommon over the precordium

In severe attacks the onset of pericarditis may be marked by vomiting, by a sharp rise of temperature and by restlessness, precordial pain and tenderness may occur As the heart swells in the severe attack breathlessness increases, the veins of the neck become engorged, and other signs of failure with congestion appear The enlargement of the liver is often conspicuous oedema of the feet is unusual or it is slight When the heart is greatly enlarged, owing to its dilatation, or to dilatation with pericardial effusion, signs of compression of the lung appear below the angle of the left scapula The failure is not due to valvular defects, for it appears when these are undeveloped, neither can present-day histological methods reveal changes in the muscle that adequately account for it The failure comes, and it goes, hand in hand with other symptoms and signs of intoxication, and is to be attributed to poisoning of the muscle.

The rhythm of the heart may be disturbed by the occasional occurrence of partial heart block or extrasystoles

Nodules are seen clinically in the subcutaneous tissues in about a tenth of the cases, and especially in those running a very prolonged and severe course They occur chiefly over bony points such as the elbow, knuckles, knee ankle, spines of vertebrae, and cranium, but also on the tendons and tendon sheaths of hands and feet There may be scores of them, but more often they are few in number and must be searched for closely Large nodules, several centimetres in diameter, are easy to detect, but those of a few millimetres, which are much commoner are apt to escape attention They are best displayed by putting the skin on the stretch, when the nodules lift and blanch the skin The skin moves easily over them they are painless and firm or almost hard to the touch, and are attached to the deeper tissues though usually slightly movable The larger masses are often aggregates of smaller ones Nodules appear quickly, and last a few days or weeks

Sweat rashes are common, of the pronounced skin eruptions, erythema multiforme is the most frequent purpura is occasional

Chorea —The nervous syndrome, with its mental disturbances, muscular weakness and inco ordinate movements, will not be described here Occurring alone it runs a course of six weeks or longer, which is almost always without fever The heart is much less frequently involved in this type of malady than when there are arthritic changes Carditis occurs, however, and, though usually mild, is sometimes severe

COURSE AND PROGNOSIS

Death may occur in acute and very severe cases, but these are rare A first attack is rarely fatal When cardiac dilatation has been slight and an apical systolic murmur has developed, these signs disappear more often than not as a first attack subsides But the disease is one that has a strong tendency to recur or to present relapses, and when the original manifestations of cardiac involvement are more serious, or when the signs reappear in a recrudescence of activity, evidence of residual damage is more frequent and permanent It is not clear that the heart becomes quite free from active disease during periods of symptomatic quiescence, and that there is not still a smouldering inflammation, but it does seem beyond doubt that the main mischief is usually done by bursts of more acute inflammation that are recognisable at the time Thus, while recovery

of normal cardiac signs is frequent after a single febrile illness it is unusual after a second and is very rare after a third, the statement being true if the attacks are separated by long or comparatively short periods of time Each attack may leave the heart noticeably changed for the worse The amount of cardiac involvement is related to the length of the active clinical course, and the cases, and they are many, that run from one relapse to another for periods as long as six months or even a year or more emerge from the illness, if they survive it, in a permanently crippled state Even so there is no immunity, further infection slight or grave being the rule

The development of mitral stenosis is gradual the signs becoming clear in general after a number of distinct illnesses or after very long drawn periods of recrudescence Among the early signs are the harsh systolic murmur, the appearance of a sound in mid diastole, giving a sort of gallop rhythm The extra sound is probably an accentuation of the normal 3rd heart sound, often occurring at this phase of the cycle and especially to be heard in children Later a diastolic murmur appears at the same phase of diastole or runs through diastole as it does characteristically in mitral stenosis of the adult Another slow development is adherency of the heart to the chest wall in cases where pericardial inflammation has not confined itself to obliterating the sac but has spread beyond it

The description of rheumatic heart disease as it develops to become chronic and as it is here described in outline is incomplete in two particulars On the one hand, it is clear that the heart may also be affected permanently by rheumatic fever occurring in adult life, and manifesting itself as a more acute attack in which arthritis is prominent though in this form the heart usually escapes On the other hand the past rheumatic illness cannot always be identified In cases of mitral stenosis in adults a history of infection is obtained in about half the cases only this is due in part to reliance upon a history of an illness with arthritic symptoms It is due also to the development of serious heart disease as an insidious rheumatic process, unaccompanied by such symptoms as would be regarded by patient or patient's friends as constituting definite illness The more closely children who have displayed single and definite attacks of rheumatism are watched, the more evident is the difficulty of stating that there is no longer active mischief Without presenting fever the child from time to time is unfit and has perhaps pains in the limbs, the pulse rate is at times unduly rapid, the routine

examination brings to light a few days of slight temperature, a friction rub, a temporary prolongation of the A-V conduction interval, a dropped beat, or other sign that but for close and frequent scrutiny would escape notice Similar incidents are to be noted in young patients with mitral disease who give no history of past illness Another indication of stealthy invasion is the not infrequent discovery of unsuspected active lesions of rheumatic type in the hearts of patients dying of cardiac failure

The mortality during the active stages of the disease is usually estimated at about 5 per cent in the first year, and 20 per cent in the first ten years, after the onset The rate varies much, however, and is very high among slum children who fail to obtain early care and treatment It is variously reckoned that of those developing rheumatism in childhood, 60 to 80 per cent ultimately manifest clear signs of heart disease.

The prognosis for the individual attack of active mischief depends upon the general strength of the child, the appearance of debility and anaemia being unfavourable, it depends especially on the signs in the heart and particularly upon the degree of failure present and *it depends upon the course the infection is taking, and that is often* impossible to estimate A high, brief fever that has been falling is more favourable than a lower fever long continuing A first attack is more favourable than a second, a second than a third, as already indicated

The distant future of the individual case depends upon when the injury to the heart by active rheumatism will end, and the extent of mischief that will then prevail To prophesy at all accurately would require not only precise knowledge of the damage already present, but foresight of the time and severity of reinfection The prognosis from this standpoint will be greatly simplified when we are able to prevent the recurrence of active mischief It is manifest from experience that patients who are properly guarded are in a much more favourable position than those who are not *It is also in general clear* that as each year passes and the child is growing towards adolescence and maturity, the most dangerous period is passing The outlook is very unfavourable in the child continuing in a crowded and unhygienic home, and maintaining in these circumstances signs of ill health and presenting frequent recurrences of low grade infection The *outlook is* unfavourable in cases presenting subcutaneous nodules.

TREATMENT

The patient in whom active disease exists should be removed from the unhygienic home to an institution with proper nursing facilities Rest is essential until active mischief is no longer suspected, namely, until temperature and pulse rate are continuously normal and have been so for several or, in the case of long illness, many weeks An object of rest is to avoid strains of joints or increased work of heart that may help to light up active trouble Tender joints should be swathed in cotton wool Patients who are febrile and sweating should be dressed in flannel or woollen clothing that is easily removed and food should be soft and much of it taken in the form of fluid Fever is treated by means of sodium salicylate or the acetyl compound, both fever and pain are abated by these remedies, though they appear not to influence the course of the disease otherwise Pyramidon, being less toxic than salicylic acid derivatives, is also used, but being less effective in lowering fever is more suited to the treatment of chorea The pain of pericarditis and restlessness are treated with opiates The use of digitalis for failure with congestion in this or any other acute febrile illness, is not usually to be recommended

Patients in whom the disease is running a long course should be moved whenever possible to a hot sunny climate The benefits of this procedure are rapidly displayed and signs of infection often completely disappear Experience is beginning to show, too, that recurrences are rare so long as the child is kept in tropical or subtropical climates In temperate climates these conditions should be reproduced as far as possible by keeping warm those patients who are fit to be treated on couch or on chair, and by exposing them to air and sunlight

Convalescence should proceed slowly and be well consolidated, in cases of carditis the end of active infection is most difficult to gauge, and months must pass before recovery can be regarded as complete Return to exercise must be very gradual Tonsils that are obviously diseased, or frequently become infected, should be removed, but routine tonsillectomy has not been shown to reduce the frequency of reinfection.

CHAPTER XXII

CHRONIC RHEUMATIC HEART DISEASE

In northern climates rheumatism *accounts for as much chronic heart* disease as any other single agent, and probably for not less than 30 to 40 per cent of all cases This percentage varies greatly for different age periods *In children and in adolescents, once* we exclude congenital malformations, it is the only important cause When a cause can be assigned, it is rheumatism in the great majority of *patients in the third and fourth decades of life* After the age of forty syphilis begins to take a place After fifty is passed, arterio sclerosis assumes an ever increasing importance, till it dominates *heavily*

A description of the established lesions, or chronic forms of disease arising out of rheumatic infections, has been anticipated by the account of their development and by the preliminary descriptions of structural lesions and functional disturbances of the earlier chap ters of this book In this section, therefore it will be unnecessary *to do more than outline the chief manifestations of the chronic* rheumatic heart

The patients may display valve defects, signs of cardiac enlarge ment, *symptoms and signs of failure, irregular heart action,* etc *It* will be profitable to note the frequency with which many of these phenomena appear, and to examine, where it is useful to do so, their interrelationships, with a view to obtaining *a simple basis for* subse quent grouping

Chief Manifestations

Valve disease —The frequency with which the valves are affected in the active stage of the disease, and the measure *in which they* become deformed in healing, have been described (pages 131, 140, and 199) As might be expected from their development, cases of rheumatic

heart disease almost always display clear clinical evidence of valve disease, that is to say, they show signs of mitral stenosis, aortic regurgitation, or aortic stenosis Mitral stenosis is twice as common as aortic regurgitation and twenty times as common as aortic stenosis, aortic and mitral disease are not infrequently combined Adult cases in which there are definite evidences of disease, such as enlargement of the heart, or fibrillation of the auricles, or congestion, without one of the named valve defects, are comparatively rare This statement is made subject to the understanding that every care has been taken to recognise early cases of mitral stenosis (page 143) Rheumatic patients in whom there are no signs of cardiac enlargement and no evidence other than a systolic apical murmur are numerous, these have been referred to at an earlier stage (page 156) Here it may be well to comment again upon the significance of the systolic murmur in its present relation to rheumatic disease First it may be said that such a systolic murmur appearing during the period of a rheumatic infection may form a guide to cardiac involvement (page 201), but experience shows that it has far less value as a sign left behind when active mischief has disappeared A heart that is left with this sign only is one which at the most has been lightly involved, and there is actually no evidence to show that, taken together with the history of rheumatism, it has appreciably more significance than has rheumatism by itself

Pericardial adhesions —Many of these cases show after death simple obliteration of the pericardial sac, a condition, however, of little consequence

Enlargement —In an earlier chapter hypertrophy of the heart and of the right and left ventricles have been discussed in relation to the chief valve defects (page 117) In rheumatic heart disease signs of conspicuous enlargement are encountered chiefly in cases of aortic regurgitation, and usually but not always this is free regurgitation, comparable signs are found also in a few patients suffering from mitral stenosis, though in general the hearts in these present less evidence of enlargement Occasionally cases are seen in which conspicuous enlargement occurs in the absence of any sign of valve lesion or pericardial adhesion The signs of cardiac enlargement vary very greatly from case to case, as judged clinically, hearts of almost any size may be found in association with any of the valve defects Enlargement in its various degrees runs more parallel with various degrees of failure than with the estimated and varying burdens of work.

Breathlessness and failure —Breathlessness on effort is the chief symptom of rheumatic heart cases There are patients in whom exercise tolerance is good, who nevertheless present aortic regurgitation or mitral stenosis, and may or may not have signs of some enlargement of the heart, their number is limited Chronic rheumatic heart cases show all grades of defective exercise tolerance, it is the symptom that ultimately becomes linked with signs of failure as the reserves are exhausted

There is a general but imperfect relation between exercise tolerance and the size of the heart Good exercise tolerance is almost confined to patients with little or no cardiac enlargement Those with fair exercise tolerance mostly present definite or moderate, rarely considerable, enlargement Patients with considerable enlargement are usually breathless with little or no effort, for the most part they show systemic and pulmonary congestion or are near to doing so Patients with congestion usually exhibit unequivocal signs of enlargement The relation between failure and size, however, is not a strict one thus it is broken by patients who, while having small hearts are easily rendered breathless The most notable of these are instances of mitral stenosis, breathlessness is more conspicuous in association with mitral stenosis than with aortic regurgitation There are instances, too, of deep venous engorgement in patients who show few signs of enlargement Relatively small hearts fail, though not so frequently as do large ones A small heart and systemic congestion that is deep and long continued is an association suggesting encasement of the heart by fibrous bands or calcified pericardium (page 181)

Congestion, predominantly systemic, is the usual termination in cases of rheumatic heart disease Failure is due in part to the unusual burdens of work that the heart has to bear, such as valve defects of various kinds, and sometimes strong adhesions, for these deplete reserves But the chief factor, nevertheless, must be inherent in the muscle itself, for failure can occur without the increased burden And yet muscle that has failed cannot at present be recognised histologically or biochemically There is increase of the interstitial fibrous tissue, a little atrophy here and there, a little fatty change, for the most part the fibres seem sound, and the muscle presents no more change than is often found in hearts that have not failed The change in the quality of the muscle is more subtle, and frequently it is brought about by some recent infection from which the patient has suffered Not only a rheumatic infection, but infections of other

types, or infarction, endanger or terminate the lives of those who are near to congestion or actually display it

Anginal pain is experienced by some, these, if young, are nearly always cases of free aortic regurgitation with much cardiac enlargement Cases of auricular fibrillation or of mitral stenosis rarely suffer from angina, but a few of the latter and a few cases of rheumatic heart disease without valvular trouble experience angina after passing the middle years of life

Precordial ache, soreness, or other discomfort is a frequent minor

Auricular fibrillation is one of the most serious consequences of rheumatic infection and plays an important part in the course of chronic rheumatic heart disease That the relation to rheumatic disease is a very close one is shown by the fact that about two thirds of all cases of fibrillation belong to this group Among mitral stenosis cases, and also among rheumatic cases that present no definite evidence of valve lesion, seen in adult outpatient departments about a fifth exhibit auricular fibrillation Aortic cases present it much less commonly

Apart from the past rheumatic history the reason for the frequent association of mitral stenosis and fibrillation is unknown It would be incorrect to regard it as caused by mechanical strain in the auricle, for it can occur when the auricular muscle is hypertrophied or when it is largely atrophied It cannot be ascribed with any confidence to such lesions as occur in the auricle, the muscle of which usually shows a good deal of fibrotic, atrophic, and fatty change, but not constantly There is no known mechanical strain or muscular lesion in the cases of fibrillation which is not known to exist in cases of normal rhythm also The ultimate cause of fibrillation is unknown

Auricular fibrillation is prominent during a relatively late phase of rheumatic heart disease It may occur at any time after infection, from the acute stage onwards, but its incidence begins to be heavy at about twenty five years and, as the disorder of mechanism is essentially persistent and fresh cases of it develop, the proportion of cases showing fibrillation mounts steadily with age, at least half the cases of mitral stenosis reaching forty years are affected by it Owing to its very frequent association with mitral stenosis, and to its baneful effects, the onset of fibrillation makes a chief event in

the lives of a large proportion of these patients There may be a preliminary period of paroxysmal fibrillation, but more usually fibrillation comes and, having come stays to distinguish the last phases of the disease

The effect of fibrillation upon the circulation has been considered (page 93) In *rheumatic heart disease, fibrillation is usually associated* with a high rate of ventricular response, owing probably to relative health of the junctional tissues When it comes it therefore makes a heavy call upon the heart s reserves, and, as these are generally reduced, and often much reduced already it almost always occasions much breathlessness, and often and quickly brings obvious congestion Failure with congestion, associated with auricular fibrillation, is the usual terminal picture, and the end in most cases would come quickly were it not for digitalis

Paroxysmal tachycardia and auricular flutter —The first of these is common and the latter is much rarer They both favour cases of *mitral stenosis with cardiac enlargement* and at their onset have much the same effects on the circulation as has fibrillation of the auricles

Embolic and thrombotic accidents —Embolism giving rise to infarction of the lung has been described fully (page 23) Simple emboli arise in similar circumstances from the left heart and find their way into the systemic system The case is usually one of mitral stenosis and auricular fibrillation the clot is formed in and detached *from the left auricular appendix Sometimes the emboli are derived* from the apex of the left ventricle The accident may happen when normal rhythm is restored by quinidine (page 101) These uninfected emboli may lodge like infected ones (page 191) in any artery, but the most important is the middle cerebral

Very occasionally in mitral stenosis a large thrombus becomes detached within the left auricle It lies loose as a ball in this chamber and may cause sudden death by plugging the mitral orifice

Hemiplegia, with or without aphasia, is not infrequent as a sudden accident in mitral stenosis with fibrillation and in other forms of rheumatic heart disease In these it is usually embolic Hemiplegia occurs, of course, in other cardiovascular cases, thus it is not infrequent in arteriosclerosis and hypertension but in these it more often results from thrombosis or haemorrhage Whether it arises from embolism or thrombosis, partial or considerable recovery is the rule, occasionally complete recovery happens within a few hours or days Treatment of the paralysed limbs by massage and proper

posture should begin after the first few days, if the patient's condition permits, and should be continued for a few weeks after improvement has ceased to be perceptible

Thrombosis of veins occurs chiefly in cases of failure with congestion, the most frequent sites noted clinically being the veins of an arm or leg The obstruction causes cyanosis and oedema, the latter often conspicuous, in the corresponding limb, the temperature of which is often distinctly raised The affected limb should be protected and kept at absolute rest for several weeks

Acute pulmonary oedema, an occasional accident in mitral stenosis, and a less frequent one in aortic disease, has been described already (page 22)

Outline of Types, Treatment, and Prognosis

As has been seen, cases of chronic rheumatic heart disease present signs of valve disease of different forms, of cardiac enlargement in different degrees, of disordered rhythm of different kinds, and symptoms and signs of failure in different amounts These do not group themselves simply, but combine in a very large number of ways, so that it would be difficult to find many patients exactly alike In attempts to classify, one way would be to use valve defects as the basis, this is the time worn plan, it has never been very serviceable either from a clinical or pathological point of view A classification by valve lesion is too anatomical, too little expresses functional [illegible]

division uses neither an anatomical nor a simple functional basis, it adopts a natural classification based upon clinical experience The groups here specified are not entirely comprehensive, neither are they sharply separable, but they enable the display of all the common forms that chronic rheumatic heart disease assumes, and they subdivide the patients into a few simple and useful categories from the standpoint of prognosis and treatment

(*a*) *Valvular disease the chief finding*—A not inconsiderable number of patients present signs of valvular disease, usually a slight aortic regurgitation or a mitral stenosis, but little or no evidence of enlargement of the heart, and an exercise tolerance that is good or fair They are cases that have come under observation for minor symptoms, or in which physical signs of valvular disease are

discovered unexpectedly The outlook for these patients is in general good, most of them will live for very many years, and live busy and even active lives, taking exercise such as walking, riding, swimming, cycling, golf, or engaging in manual work, such as carpentering, fitting, plumbing, decorating Many men of this class fought in the trenches in the 1914 war The women do house work, or in factories such work as packing or machine work, many of them marry and bear children without harm (page 274) The patients require little management Little should be said to them about their hearts, they need occasional advice about their work and habits

(b) *Auricular fibrillation the chief finding*—The cases of this group come complaining, when untreated, of palpitation, of breathlessness with brisk walking exercise, present few or no signs of cardiac enlargement, and are almost always, but not always, found to display, in addition to auricular fibrillation, signs of mitral stenosis The ventricular rate is controlled by digitalis Although it is remarkable how much many such patients do with impunity, the outlook in them is not so good, and life should not be so strenuous as in the preceding class

(c) *The central and chief group*—These patients complain chiefly of breathlessness when they walk on the flat, tolerance of exercise being only fair or decidedly poor, the heart is found to be definitely or moderately enlarged, and signs of aortic disease or mitral stenosis, or both, are discovered In those with mitral stenosis, auricular fibrillation is often present

This is the largest class of rheumatic heart case in adults The prognosis is not good, there is a mortality of one third of the cases within ten years of diagnosis Death when it comes is from cardiac failure in the majority This is especially the case for mitral stenosis Infective endocarditis claims some of the cases Many of the group survive longer, however, and, surviving these years, show for the most part little change In this group the death rate is equally great for mitral stenosis, for aortic regurgitation, and for a combination of the two Factors on which chief stress is to be laid are the degree of cardiac enlargement and the cardiac reserves The patients lead, or should lead, very restricted lives, being unfit for more than light or sedentary work, or for any active exercise, the prospect of living is lengthened by living carefully It is unwise of women in this class to bear children In those who display fibrillation of the auricles, digitalis is to be used appropriately All require close supervision, their symptoms should be a matter for frequent

interrogation and test, the veins of the neck and the liver should be watched most carefully As years go by, a number develop signs of failure and usually do so abruptly Most of the examples of infarc tion of the lung and of hemiplegia that are encountered come from this group

(*d*) *Cardiac enlargement the chief finding* —There are instances of considerable enlargement of the heart, without actual congestion, occurring chiefly in conjunction with free aortic regurgitation, but occasionally found apart from it Almost without exception the cases have poor exercise tolerance, being breathless upon the slightest exertion Among them also are cases of severe angina pectoris at rest, which have been described earlier (page 65) The patients rightly spend much or all of their time in bed, the prospect of life is but a few years, and is very short if the subjects are not well guarded

(*e*) *Venous congestion the chief feature* —When present, systemic venous congestion dominates the picture, controlling the outlook and governing treatment, valve lesion, if present, is but a subsidiary consideration This congestion may be combined with aortic disease or with mitral stenosis the latter is the more frequent combination, there may be no definite valve lesion to diagnose It matters little Congestion is associated with various degrees of enlargement, usually moderate or considerable Because it reacts well to digitalis, the case of congestion with fibrillation is more favourable than a similar case displaying normal rhythm Chronic systemic congestion or persistent pulmonary congestion that cannot be relieved are manifestly and immediately threatening Cases of congestion, even when responding to treatment customarily relapse, and the prospect here is not in general long though it varies by some years Those patients who are relieved slowly, and those who, when relieved, present moderate or considerable enlargement of the heart, are least likely to survive

This subdivision of chronic heart disease into a few appropriate categories sufficiently indicates the chief lines of treatment to be followed, as these are described in other parts of this book, and it begins to outline a plan of prognosis that proves satisfactory in practice, and which is dealt with more broadly in the final chapter In rheumatic heart cases that have become established the heaviest death rate is between the fifteenth and thirtieth years and it is heaviest in the early years of this period because these are the years of active disease By the fortieth year less than half the patients

survive This statistical statement has only a limited value, however, in assessing the prospects of individual patients, a number of whom live on to old age Individual prospects are to be gauged on the lines laid down in the preceding classification

INFECTION

In so far as rheumatic infection is concerned, enough has been said in the preceding chapter to make it clear that it brings an element of great uncertainty into prognosis In dealing with young patients, *still in their teens or entering their twenties, the possibility* of present rheumatic infection must still be considered but as the patient is older, so search for and exclusion of signs of this infection becomes less important The occurrence of subacute bacterial endocarditis cannot be foreseen A proportion of these chronic rheumatic heart cases *acquire this infection* and, *when it is* diagnosed, prognosis changes correspondingly, but as its coming is unpredictable and is infrequent, it should not enter into the consideration of the prognosis of the uninfected individual

DEGENERATIVE CHANGES

Patients who survive well into and beyond the forties enter the phase of life when degenerative changes appear Arterial disease begins to take a prominent place, perhaps the coronary arteries become sclerosed and bring in an anginal syndrome, perhaps the arterial pressure becomes raised Breathlessness becomes a more severe and persistent symptom These new troubles, not infrequent in late rheumatic heart disease, bring fresh complexity to the case

CHAPTER XXIII

SYPHILIS OF HEART AND AORTA

AMONG white people of northern climates, syphilis is a declining cause of chronic disease of the heart and vessels It has always been less important than rheumatic fever and arterial disease It attacks particularly the aorta, involving its valves in the later years of life It is much more frequent in males than females It is the chief cause of aortic regurgitation in old men

Syphilitic aortic disease, like tabes dorsalis is a very late manifestation of the original infection, it is the most frequent lethal consequence of this infection The reason for the long latent period is unknown, but it averages about twenty years infection occurs usually in the twenties and the peak in the incidence of syphilitic aortitis is near the forty fifth to fiftieth years It may follow congenital infection, and then the latent period is in general shorter

PATHOLOGICAL ANATOMY

In attacking the blood vessels, syphilis concentrates upon the aorta at its origin, the ascending and the transverse aorta is usually involved in decreasing degree, as it is traced onwards to the mouths of its brachiocephalic branches Less commonly the process invades the descending aorta, occasionally the abdominal aorta The mouths of vessels issuing from a diseased aorta are always involved, but the branches of the aorta in their length escape, the chief though infrequent exception being the innominate artery The disease often affects the sinuses of Valsalva heavily, here the mouths of the coronary arteries are implicated, the disease does not invade the vessels themselves, but ends abruptly at their origins

The disease consists of a chronic inflammatory process, marked

by lymphocytic infiltration and fibroblastic reaction, and by intimal thickening of local vasa vasorum It probably starts in the adventitia, it invades the wide media, breaking and separating its elastic laminae Intimal thickening occurs, and the process of repair continues to lay down fibrous tissue here and also in and around the weakened aortic wall, while the inflammation continues The internal surface of the aorta is characteristically deformed by numerous, large and small, often depressed, fibrous scars, of white or pink colour Atheromatous changes are mixed with these in a degree increasing with the age of the subject The mouths of vessels issuing from the diseased aortic wall are much fibrosed and, as shrinkage in these is less opposed by dilatation, they are often greatly constricted, a matter of supreme consequence in the instance of the coronary vessels, and of diagnostic import in the case of larger branches

As the affected wall of the aorta becomes deficient in resisting elastic laminae, it stretches under the pressure of its contained blood It may dilate more or less uniformly until its diameter is at least twice that of a normal aorta, at a time not sharply distinguished in the process, or by terminology, dilatation becomes fusiform aneurysm" Expansion may proceed less regularly A dilatation in one direction may become a definite bulge, this may widen to form a small round projection, or saccular aneurysm, or one bulge may unite with others to form a much larger and irregular aneurysm Aneurysms vary much in shape, size, position, and in their direction of pointing All structures that lie in the path of expansion are compressed, distorted, displaced, or destroyed Bone of rib or vertebra and other resistant structures are eroded Saccular aneurysms are often lined, and sometimes filled, by laminated clot This may be dense and solid, or pierced by blood channels

The disease attacks the aortic valve through its commissures, which become inflamed, the cusps become thickened and retracted Simultaneously, in greater or less degree, the structures supporting the valve are weakened, the valve ring dilates, and the cusps, lengthened but shrunken, are held away from the orifice, which they were set to guard

Hypertrophy of the heart is the rule in syphilitic aortitis, it is conspicuous in cases in which aortic regurgitation has been free, and then affects the left ventricle in general more than the right Gummata are occasional at an earlier period of the infection and sometimes invade the auriculoventricular bundle (page 105) But in

the later stages active inflammatory lesions are small and rare An increase of interstitial fibrous tissue is found, but changes in the muscle fibre are not pronounced

In dealing with the clinical manifestations of syphilis of the heart and aorta, it will be convenient to consider the aorta first and the whole condition subsequently

Dilatation and Aneurysm of Aorta

SYMPTOMS

The complaints of patients suffering from dilatation or aneurysm of the thoracic aorta are extremely varied By far the most frequent are pain and breathlessness But, as the aorta is syphilitic, the coronary mouths often narrowed, and cardiac failure is present in greater or less degree, these symptoms cannot be ascribed usually either to dilatation or to aneurysm

Pain across the upper part of the chest may be frankly anginal often it is less characteristic and then, while sometimes to be ascribed to erosion of the sternum its cause is more frequently obscure Whether or not pain can be produced by the stretching or inflamma tion of nerves lying in the wall of an enlarged aorta is unknown

Breathlessness is usually the result of cardiac failure, but it may be contributed to, or caused by, compression of trachea or bronchus or by compression of the lung and in other ways as well

Most of the important symptoms of aneurysm are definitely pro duced by pressure on other structures Thus pain comes from irrita tion of nerve roots, or from the erosion of bone, difficulty in swallow ing comes from pressure on the gullet Haemoptysis comes from erosion and ulceration of the trachea, bronchus or lung an actual leak from the aneurysmal sac itself being not unusually demon strable Cough may result from the same lesions, or from infection of air passages or lung These and other pressure symptoms will be noted again a little later

LOCAL SIGNS

Pulsations —Several forms of pulsation occur in cases of aneurysm and form valuable signs

Pulsation in the 2nd right intercostal space, near the sternum, is the most frequent early sign of aneurysm of the ascending aorta The sign occurs, however, much more commonly when the right lung retracts from the base of the heart, or when the heart is over-

acting especially in aortic cases with or without aortic dilatation But in these instances pulsation is more visible than palpable, and it is a brief and not a sustained systolic movement as is that of aneurysm When due to aneurysm the area that pulsates is always dull to percussion, but rarely so in simple dilatation of the aorta

Pulsation is also caused by aneurysm in the 2nd or 3rd left inter costal space near the sternum, though less frequently Local pulsation in this region much more usually results, however, from retraction of the left lung, dilatation of the pulmonary artery, or upward dis placement of heart when the contents of the abdomen are greatly increased Pulsation in the same spaces is common as part of a diffuse pulsation over the heart (page 120) In aneurysm alone is there a sustained systolic pulsation, dulness, however, may also accompany a dilated pulmonary artery

Movement of the manubrium sterni, if systolic and sustained, is an almost diagnostic sign of aneurysm and is not infrequent

Local pulsation from aneurysm may appear on occasion in almost any other part of the chest, front or back, and may affect rib or space, but it is infrequent in the back and on the front of the chest below the 4th rib

Episternal pulsation, systolic in time, is frequent both in aneurysm and dilatation of the aorta When the arch of the aorta enlarges up wards appreciably, the manubrium tends to be displaced forwards, sometimes in aneurysms it is obvious that the trachea and sternum have become unusually separated, whether this is so or not, a finger pressed down behind the sternum may feel the top of a dilated or aneurysmal aorta

Pulsating tumour of the chest wall is a late sign of aneurysm An aneurysm may break through the chest wall, eroding sternum, ribs, or cartilages in the process at any of the places named in the last paragraphs, the site of election is the base of the heart, and especially to the right of the sternum Such tumours bulge at first a little, then become hemispherical or conical as they grow They are tender, and the skin covering them becomes inflamed They are recognised for what they are almost always without any difficulty, since their form and site are characteristic, and because they pulsate The pulsation is systolic and sustained, it is expansile—that is to say, it takes place at right angles to each part of the tumour's surface Expansile pulsa tion is not to be expected if the tumour is only just raised above the level of the surrounding skin It becomes obvious or conspicuous

when the tumour projects obviously or conspicuously Thus the expansile quality of pulsation in these tumours is not very helpful, because their nature is already evident before the sign is obtained Very uncommon neoplasms of the chest wall may pulsate slightly and in an expansile fashion, but the circumstances in which these occur are so different from those of aneurysm that they rarely should be confused Pulsating empyema is easy to recognise, occurring usually near the left nipple, its pulsation is feeble and the fluid contained in the hernial projection of pleura returns to the main pleural sac under the light pressure of the hand

Tracheal tug —The aorta arches over the root of the left lung and, when pulsating unusually, forces this root down and tugs on the trachea The tug is felt if the tips of the two index fingers are placed under the lower margin of the cricoid cartilage and the cartilage is gently lifted headwards The tug when vigorous is diagnostic of aneurysm but a slight, and distinct, is also encountered in some

must not be confused with the forward impulse ...
beating carotid arteries

Dulness —Dilatation of the aorta usually produces dulness behind the sternum at the level of the 2nd and often up to the 1st interspace The sign may be only just detectable or distinct The borders of dulness may extend a half inch to the left and a little more to the right of the sternal borders An area of dulness exceeding 3 inches (8 cm) in diameter rarely results from simple dilatation ... aneurysm becomes more ...

Heart sounds —Because enlargement ... to and fro murmurs generally accompanied by aortic regurgitation, to and fro murmurs are usually audible at the base of the heart When aortic regurgitation is absent or is slight, a systolic murmur at the base (with or without thrill) is usual An accentuation of the 2nd aortic sound is frequent the accentuation is peculiar in having a slight musical quality the noise is reproduced when a slack strip of linen is thrown into sudden tension Though not diagnostic it is a useful ... rise to sign to aortic dilatation, and is frequently as ...

palpable 2nd sound, an equally suggestive sign termed "diastolic shock"

The heart sounds are often remarkably well conducted to those parts of the chest wall towards which large aneurysms are projecting

X-ray shadow—Much of the contour of the aorta can be determined by X ray exploration In normal adults the right border of the cardiac silhouette is composed of two parts—namely, the rounded outline of the right auricle below and the almost straight shadow of the superior vena cava extending from this to the clavicle The transverse aorta as it passes backwards through the mediastinum appears as a semicircular projection, or knob, forming the top of the left border of the silhouette The first part of the shadow below this knob may correspond to the beginning of the descending aorta or to the pulmonary artery, and is usually little curved The left border is completed by the shadow of the ventricle These points are displayed with sufficient clarity in the orthodiagram shown in Fig 21, page 119 It is usually possible to obtain a clear idea of the contours of the ascending and transverse aorta by screening The contours become easier to decipher in their whole length as the aorta becomes enlarged, and information as to the size and shape of its several parts is increased by rotating the patient into a right or left oblique position Many X ray measurements have been devised with the idea of expressing the size of the aorta in numerical terms, but most of these are subject to error or misinterpretation, and they do not add appreciably to a simple judgment of the form and area of the vessel

When the aorta is dilated, the density of its shadow is increased, its curves become fuller, but they retain their smoothness, the ascending aorta takes a more prominent part in the right outline, the knob is outlined by a fuller curve, the knob may be raised until it meets the clavicle it extends farther to the left Often the descending aorta is visible and descends for some distance before it is lost in the left border of the heart's silhouette Thus the shadow of the basal vessels is widened at all levels (Figs 33 and 34)

Aneurysms of the aorta present very diverse X ray pictures Early saccular aneurysms are usually recognised by closely following the visible contours of the aorta in appropriate planes, so as to detect at i disturbances of its normally continuous and rounded curves, tion isrection of the line changes more or less abruptly where an level ot m joins the aorta In oblique radiograms or screening the

contours are examined for parallelism, departure from the cylindrical form is significant When a definite tumour is present, its shadow, if it is aneurysmal, must blend with, and present a similar density to, that of the remainder of the aortic silhouette Aneurysms (Figs 35 to 38) present sharply defined margins, for the most part smooth contours, and no irregularities of density Sharply defined circular outlines are particularly suggestive, as they can only be confused with cysts The presence or absence of pulsation at the border of the shadow is rarely helpful, because aneurysms often fail to display it, because the normal aorta may be displaced by a solid tumour, and because tumours often have pulsation transmitted to them Expansile pulsation in a round tumour is of course decisive, but this is not often seen X ray signs in trachea and oesophagus are described later

SYMPTOMS AND SIGNS OF PRESSURE

Owing to the anatomical relations of the aorta to the mediastinum, aneurysm of this vessel frequently causes symptoms and signs by exerting pressure upon structures surrounding it Pressure manifestations are usually the result of aneurysm of the transverse aorta, and the chief are as follows —

The oesophagus is one of the most frequent structures to be affected, many patients complain of difficulty in swallowing solid food Very few are unable to swallow and few regurgitate, a point of importance in differentiating from other forms of stricture and diverticulum If a mouthful of stiff barium paste is swallowed while the patient is being screened an actual obstruction to the oesophagus is readily demonstrable The obstruction is usually level with the top of the arch of the aorta or just below it A noticeable lateral bend or displacement of the oesophagus to the right may be noted when the patient faces the screen, but this is usually more distinct when the subject turns half left with the right shoulder forward The arch of the aorta normally indents the outline of the barium filled oesophagus in dilatation or aneurysm of aorta this indentation is increased and shows especially in right oblique and antero posterior views

Compression of the lung by aneurysm is uncommon, but occurring results in local dulness, deficient air entry, and crepitations Occasionally the lung is eroded by the pressure, giving rise to haemoptysis

Pressure on the trachea may be responsible for breathlessness or stridor and, when the aneurysm ulcerates the trachea, for cough

with blood streaked sputum, or heavier haemorrhage The most frequent sign in the trachea is its slight but distinct displacement to the right at the root of the neck, this may be detected at the bedside, and is well seen in many of the radiograms Deformity of the trachea is responsible also for metallic (brassy) cough

Pressure on the left bronchus is common and causes at first deficient air entry at the left base characteristically producing absent breath sounds over lung that is resonant to percussion later collapse, bronchiectasis and local pulmonary infections may follow

Compression of the left recurrent laryngeal nerve causes an

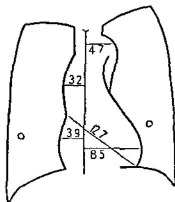


FIG 33 —Orthodiagram Man aged 47 weight 129 lb Aortic regurgitation (free) syphilitic aortitis general dilatation and *elongation of the aorta The ascending* transverse and descending parts of the aorta are all clearly visible Measurements in centimetres

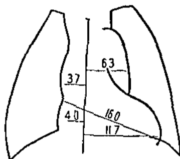


FIG 34 —Orthodiagram Man aged 60 weight 159 lb Aortic regurgitation (slight) syphilitic aortitis systolic blood pressure 2 0 mm *Great widening of the ascending* aorta and of its arch The ventricle is enlarged and lies horizontally

abductor, and later complete paralysis of the corresponding vocal cord, changing the voice, making the voice hoarse and the breathing noisy

When, very rarely, the left sympathetic nerve is caught and paralysed the palpebral fissure and the pupil are narrowed, signs that are rendered relatively more emphatic by instilling a few drops of 1 per cent cocaine into each conjunctival sac, for this dilates the pupil and renders the eyeball more prominent on the unaffected side Sympathetic paralysis causes increased warmth of the face on the same side the colour of the affected skin may be unchanged, or it may be redder or paler than that of the other side according to the state of vasomotor tone in the latter Simple inequality of the pupils in aneurysm is common but is unexplained

Obstruction of the superior cava causes swelling, most notably in the face, and high colour in and often cyanosis of, the head and neck, the veins of the neck are engorged but do not pulsate If the

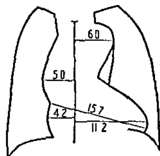


FIG 35—Orthodiagram Man aged 56 weight 151 lb Aortic regurgitation (free) syphilitic aortitis fusiform aneurysm of ascending aorta the descending aorta is also visible

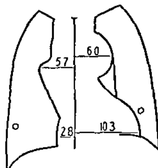


FIG 36—Orthodiagram Man aged 45 weight 139 lb Aortic regurgitation (free) syphilitic aortitis a conical aneurysm projects to the right from the ascending aorta and the transverse aorta bulges as a sac to the left in the chest

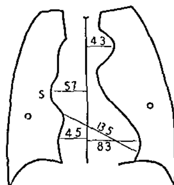


FIG 37—Orthodiagram Man aged 6? weight 167 lb A saccular aneurysm (S) of the ascending aorta bulges to the right The arch of the aorta is greatly dilated or actually aneurysmal

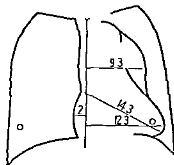


FIG 38—Orthodiagram Man aged 56. The ascending aorta and arch are dilated, and an aneurysm bulges to the left from the first part of the descending aorta.

patient survives long enough, anastomotic veins appear over the clavicle and in the region of the shoulder The blood flow in them can be traced downwards over the front of the chest or axillae Ultimately the anastomoses may form very dilated tortuous leashes

Pressure on a subclavian vein engorges the corresponding arm, and on a subclavian artery weakens the pulse beyond Occasionally

lower roots of the brachial plexus are damaged, causing neuralgia in the arm Pressure on both veins and nerve roots may yield unilateral clubbed fingers

Descending aorta —Aneurysm of the descending aorta is apt to develop very insidiously and to give neither symptom nor sign till it has grown to a large size Obstruction of the oesophagus may occur as an early isolated event Compression of lung is frequent The aneurysm erodes the bodies of the vertebrae may involve the dorsal nerve roots, and in more advanced cases may even come to press directly upon the spinal cord Erosion may be painless but a steady aching pain localised in the middle line of the back is the commonest symptom it is sometimes accompanied by local tenderness The *patient may complain when the spine is jarred and use* great caution in rising from a lying to a sitting position Pain may be more severe and radiate along the dorsal nerves Pressure on the cord produces the familiar picture of paraplegia Dulness to percussion and even pulsation may appear locally, but are rare signs A skiagram will demonstrate the state of the vertebrae and may reveal the tumour

DISTANT ARTERIAL SIGNS

Most valuable evidence of the involvement of main arteries by disease is often to be obtained from their branches Inequality in the strength of the radial pulses is frequent in aneurysm of the thoracic aorta, when found it is not to be used until traced to its source First the brachial, and then the subclavian, pulses are felt to ascertain if these participate in the inequality Inequality of the pulses at the wrist is often due to difference in the size of the two [illegible]

is the less distinct, for in that case the innominate artery is obstructed It is unusual for more than one of the main vessels issuing from the arch to be involved, though all three may be

When decreased, the pulse is anacrotic (page 138) or flat topped *in form The up stroke of the pulse is never delayed*, but, if the pulse is anacrotic on one side, then its summit is represented by the second wave of the pulse, and this gives an impression of delay There may be but a flicker in the arteries of one arm, or no pulse may be felt Weakness of the brachial pulse *is associated with a* corresponding fall of the systolic pressure reading on that side It is

erroneous to believe that diminished pulsation in the arteries necessarily means a lessened circulation in the limb, in aneurysm gross inequality of the radial pulses may be found, yet the hands may be of equal warmth, and measurement may show the blood flow in them to be equal in amount

The cause of a decreased pulse in aneurysm is obstruction at the mouth of the corresponding vessel as it leaves the sac This is usually the result of fibrosis and constriction of the mouth as part of the disease in the aortic wall, similar constrictions occur in syphilitic aortitis without aneurysm, though in these they are less common because the disease is less advanced I have known the pulses in all the three vessels to be lost in a simple syphilitic aortitis A less usual cause is blocking of the mouth of the artery by clot lying in the aneurysmal sac, it is rare for an aneurysmal sac to press upon a subclavian artery sufficiently to distort it That weakening of the pulse is materially contributed to by the wastage of its force in expanding the sac is a prevalent but erroneous notion If it were so, the femoral pulses would often show more weakness than they do

Cardioaortic Syphilis

From considering in detail the diverse effects of syphilitic aortitis, attention is turned to the disease as it affects heart and aorta together

In making its delayed attack syphilis concentrates upon the aorta Of symptoms directly referable to inflammation of the aorta none are known, a dull ache across the sternum is an early and frequent symptom, sometimes ascribed to this cause The disease usually develops far before the patient complains, and the complaint, when it comes, arises out of one of four chief events The disease (*a*) weakens the aortic wall so that this dilates or becomes aneurysmal, (*b*) invades the aortic cusps and their supporting structures, rendering the valve incompetent, (*c*) attacks the mouths of the coronary vessels and causes angina pectoris, (*d*) produces, directly or indirectly, a decline of ventricular efficiency Of these four chief results the first, with its pressure symptoms, has been considered at length The remainder have been considered at length, though not with particular reference to syphilis, in previous chapters The chief manifestations vary in their incidence or are combined in different ways But when one is displayed, others are not often long delayed

CHIEF MANIFESTATIONS

Dilatation of the aorta, distinctly recognisable, is found in at least half the cases *aneurysm* occurs in an additional group amounting to 15 or 30 per cent of the whole

Aortic regurgitation is present in the great majority of the cases, whether aneurysm is present or not When present it is almost always free, and extreme signs of regurgitation are frequent Slight regurgitation is much less common than in rheumatic heart disease, stenosis of the valve is a rarity The reason for this difference is that in syphilis the aortic ring is dilated and the cusps lose their support Rupture of an aortic cusp is usually the result of syphilis and gives rise often to a particular group of signs and symptoms already described (page 136)

Angina pectoris of unmistakable type occurs in a number of the cases The anginal subjects nearly always present aortic regurgitation Usually appearing as an angina of effort, it often reaches ultimately an unusual pitch of intensity, attacks of great severity occurring once or several times a day the attacks are frequently accompanied by a conspicuous rise of pulse rate and blood pressure (pages 65 6)

Cardiac failure —In cardioaortic syphilis breathlessness on effort is an almost invariable symptom and in a large proportion of the cases signs of congestion appear Pulmonary congestion predominates The two chief known reasons for this failure are the increased work that the heart has to do and insufficiency of its blood supply The extra work is due to the aortic reflux, and here allowance must be made not only for the advanced grades of disease in the cusps but for the increase in the size of the orifice that is to be guarded more over these patients are middle aged or elderly, and high or relatively high blood pressures are common among them Insufficient blood supply results in part from constriction of the coronary mouths and in part also from the heaviness of the ventricles that are to be supplied through them Enlargement of the heart is conspicuous or extreme in very many of these patients it is particularly so in those with aortic regurgitation Enlargement, however, and sometimes great enlargement, occurs when there is no incompetence of the aortic valve In general as in the case of rheumatic heart disease, the degree of breathlessness corresponds with the amount of enlargement Little can be said of structural change in the ventricular muscle There is a certain amount of replacement fibrosis, it is true,

but the cause of failure cannot be recognised histologically here, any more than in rheumatic heart disease Experience shows that the muscle of an enlarged heart is rarely dependable Alternation of the pulse (page 252) is not infrequent In elderly subjects atheromatous changes occur in the coronary vessels and induce secondary changes in the muscle, but coronary thrombosis is not a common accident in these patients Neither does fibrillation of the auricles often contribute to failure, failure with normal rhythm is the rule

As in other instances in which failure with congestion comes while the rhythm is normal, it is almost always progressive and terminates life within six or twelve months As in similar but non syphilitic cases, it is frequently accompanied by periodic breathing and by distressing paroxysms of breathlessness, experienced especially at night, and more closely described on page 21.

TYPES

The chief types that may be recognised conveniently are —a main group in which there is aortic regurgitation, with or without dilatation of the aorta, but no angina, a smaller group in which anginal pain dominates the clinical picture, and a third group of cases presenting aneurysm Each of these types may or may not present signs of venous congestion A fourth group is small because the cases are usually difficult to diagnose It is one in which there is syphilitic aortitis, with or without dilatation of the aorta and enlargement of the heart, but no aortic reflux It has practical importance because in it are represented many of the early cases It has theoretical interest because the patients sometimes develop cardiac enlargement, even great enlargement, and congestion, while the aortic valves still remain competent

DIAGNOSIS

In diagnosing syphilitic aortitis, it is necessary to obtain evidence of syphilitic infection, and to identify disease of the aorta The very presence of aortic regurgitation in a man of forty five years at once [illegible]

palate, and the diagnosis is in little doubt A positive Wassermann reaction or association with tabes dorsalis is more valuable still A negative Wassermann reaction, on the other hand, does not exclude syphilis as the cause

A middle aged or elderly man who presents signs of aortic re-

gurgitation should always be searched closely for the clinical signs of aortic dilatation and of aneurysm And this examination should include, whenever possible, a thorough examination of the chest under X rays For, if definite evidence of aortic dilatation is found, the diagnosis of syphilitic aortitis becomes probable, and if signs of aneurysm are found, it becomes almost certain Dilatation of the aorta, however may occasionally produce retrosternal dulness to percussion in cases of senile atheroma, especially when accompanied by high arterial tension though this is usually distinguishable by the tortuosity of the aorta under the X rays Dilatation, and occasionally aneurysm, of the aorta occur in coarctation of the aorta this condition is relatively rare and not difficult to distinguish (page 268) Aneurysm is occasional in arteriosclerosis (page 244)

The diagnosis of syphilitic aortitis when aortic regurgitation is absent is more difficult It is well to consider syphilis as the possible origin of enlargement of the heart in all patients over forty years of age in which the cause of enlargement is obscure A positive Wassermann in these, however, does not suffice, since its association may be fortuitous To make the diagnosis certain, enlargement of the aorta must be found as well

The diagnosis of aneurysm in its early stages depends mainly upon the closeness with which patients prone to display this form of aortic disease are searched, and especially upon judiciously selecting cases for X ray examination Most cases should be diagnosed before the patient complains of some symptom more or less distinctive of aneurysm, and before there are manifest signs over the front of the chest The diagnosis should hardly ever be made in patients under thirty years When a sign or symptom of pressure upon a mediastinal organ appears, it will be necessary to differentiate between aneurysm and new growth A positive Wassermann reaction will assist in this case, signs of aortic regurgitation will assist even more, because they will proclaim the aorta unhealthy, but in the absence of such assistance, the diagnosis of aneurysm will turn chiefly upon an X ray examination, upon finding that an artery leading from the aorta is obstructed, or upon the general grouping of symptoms and signs

COURSE AND PROGNOSIS

Syphilitic aortitis is a malady of later years when the natural expectation of life is not very lengthy It is a serious disease, and often comes under observation when far advanced Consequently the outlook in years is relatively short The average duration of life from the diagnosis is about six years in patients under treatment, though

quite a number live ten years or more A considerable number dies suddenly In another large group of the cases signs of failure develop, and this then progresses to end life usually within a year; a few die of acute oedema of the lungs In a small number, subacute infective endocarditis supervenes A few die of syphilitic meningitis, cerebral haemorrhage, or as a consequence of aneurysmal pressure on important structures Intercurrent maladies, including renal disease, account for many more deaths

In the individual case serious anginal symptoms and aneurysms influence the outlook unfavourably The anginal syndrome is usually severe and rapidly progressive owing to further narrowing of the mouths of the coronary vessels It is in these cases and in those of aneurysm that sudden death is so frequent In aneurysm sudden death results from rupture of the sac externally, or into trachea, bronchus, oesophagus, pleura, or pericardium The average duration in cases of aneurysm is about four years from the diagnosis When an aneurysm has bled, or by compression has caused infection in the lungs, the course will be measured in months But the prognosis is not invariably unfavourable, some aneurysms heal and in a number the condition remains unchanged for eight to ten years after the disease is discovered

Apart from these special considerations, individual prognosis turns mainly upon the degree of failure, as evidenced by symptoms and signs, and upon the size of the heart, much breathlessness, with early or late signs of venous congestion, or great enlargement of the heart, being ominous

The prognosis should take into account the rate of progress and the reaction of the patient to rest, the figures here given are for patients under full treatment and not at work There is no doubt that the prognosis is much less favourable in those who continue to do manual work, especially heavy work, which raises blood pressure and strains the unhealthy aorta

TREATMENT

The chief remedial measure in the treatment of syphilitic aortitis in all its forms is rest Manual work, unless of the lightest, should be forbidden, even in the mildest cases, and patients who present much enlargement of the heart, angina, or aneurysm should spend most or all of their time lying or sitting Diet should be light (Diet II, page 292) Strenuous acts (page 291) are to be forbidden for all time, and strictly healthy habits (page 289) inculcated Potassium iodide

(15 or 20 grains or 1 0 to 1 3 g thrice daily) has proved to be a very useful remedy in relieving pain combined with mercury in the form of grey powders or inunctions, it is used as a specific remedy and has been thought, but is *not proved, to stay the progress of the disease* In using these remedies care should be taken to avoid iodism or mercurialism

Neosalvarsan and similar preparations have been employed extensively from the same point of view, and under full courses the Wassermann reaction frequently becomes negative Ten to fifteen intravenous injections of neosalvarsan, 5 grains to test and afterwards 10 grains (0 3 and 0 6 gramme) are given at the rate of one or two doses a week and the course repeated twice each year The uncomplicated cases tolerate these injections well According to most available evidence this remedy prolongs life though this is not universally agreed Cases having angina should rarely be given this treatment, but early aneurysmal cases may

In treating thoracic aneurysm, a chief point is continuously to maintain a low average blood pressure, and this is most safely accomplished by rest by a small but adequate dietary, and by the promotion of full hours of sleep Vasodilator substances are not recommended It is not unusual to notice a decline in the size of a pointing aneurysm as blood pressure falls with rest The use of very *low calorific diets and of other remedies* such as the introduction of foreign bodies into the aneurysmal sac, having for their object the promotion of clotting in the sac, is not advised It is quite beyond our power to control the amount and position of clotting in the sac and the remedies have each *their special dangers, it is clear that clot may* form in quantity and yet fail to strengthen the aneurysm against rupture, and it is clear that clots in forming may obstruct important outgoing vessels and even the aorta itself Remedies of *this class do* more harm than good When an aneurysm is *weeping into trachea*, into oesophagus, or externally, as it sometimes does before rupturing the patient must be kept at absolute rest under morphia

Angina and failure with congestion, when present, are treated on lines already laid down

The time to treat syphilis *is during the weeks following infection* Once the disease has declared itself in the aorta the damage is usually irreparable [1]

[1] In the United States of America the death rate among *cases of syphilitic* aortitis appears *to* be heavier than in Great Britain, upon which the prognosis here given is based.

CHAPTER XXIV

ESSENTIAL HYPERTENSION

VERY high pressure in the systemic arteries occurs transiently in rare cases of suprarenal tumour It is found persistently in most cases of chronic nephritis, chronic pyelonephritis, urinary obstruction (hydronephrosis), polycystic disease, and some forms of tumour of the kidney, it occurs in basiphile pituitarism, and coarctation of the aorta

of primary inflammation of the kidneys Such renal lesions as are found are chiefly due to arterial and arteriolar disease The condition is often familial It is almost peculiar to the later periods of life, being rare before the age of forty and having its chief incidence in the period between fifty and seventy years It often follows shortly upon the climacteric in women It is not infrequently associated with obesity The immediate cause of the high blood pressure is increased tone of the peripheral vessels, and especially of the arterioles The reason why there is persistent increase of arteriolar tone is not finally known, though absorption from a damaged kidney is the probable cause

PATHOLOGICAL ANATOMY

Hypertrophy of the heart is the rule, its weight being usually double the normal Much lighter and much heavier hearts are sometimes seen The very largest hearts that occur are found when high pressure and aortic regurgitation have coexisted Although the right ventricle is usually hypertrophied too, there is no condition in which hypertrophy of the left ventricle is more dominant than in hypertension The morbid anatomy is often complicated by an association of lesions Arteriosclerosis in the aorta, or widespread in the vessels, is the rule, and the changes are often advanced, but

they are unessential to the picture and may be even less conspicuous than is usual at a corresponding age *Coronary disease is frequent* Valve disease may be associated, the aortic valves being affected by degenerative change, or by an associated syphilitic aortitis, the mitral valve being stenosed by old rheumatic inflammation Rarely the kidneys seem quite normal, they usually show changes, though these vary much in degree *The renal changes are interpreted* as resulting from thickening of the arterioles, itself largely the consequence of continuous high tension The arteriolar changes are *widespread, especially* in kidney, pancreas, and liver The kidney may be brown in colour and covered by very fine surface granulations, representing the scars of minute atrophic foci, it may be reduced in size and its surface covered by coarse granulations, the familiar and more advanced red granular kidney

Symptoms

There are no symptoms proper to high blood pressure Very many of the cases feel in good health while leading active lives mentally and physically, the condition being discovered in a routine examination conducted for insurance or other purpose Others make a variety of complaints, some of which arise from arterial disease

The onset of symptoms is usually very gradual, the patient noticing a lack of energy, fatigue, restiveness, headache, giddiness, or other minor symptoms Occasionally it is abrupt, as when severe nose bleeding, or a minor or major cerebral attack occurs

Headache is frequent, especially after exercise and particularly on waking from sleep It may be occipital or frontal and is a dull ache, or more intense and throbbing

Nervousness, irritability of temper, with or without emotional outbursts, are not unusual, especially in women who develop the condition after the menopause Failing memory and inability to concentrate are common

Minor cerebral attacks occur, these may consist of dizziness without rotation, faintness, or actual brief loss of consciousness, or they may consist of transient mental confusion or loss of memory, pareses or paraesthesiae These symptoms may be premonitory to apoplectic or other major cerebral seizures such as hemiplegia or blindness

Disturbances of vision may result from retinitis or from intraocular haemorrhages.

Cardiac symptoms are common Palpitation is frequent and may be due to forcible action of a regularly beating heart, but more usually to extrasystoles and occasionally to fibrillation The most frequent symptom belongs to cardiac failure, namely, breathlessness on effort which is mild in the early cases and increases until obvious congestion comes A little swelling of the feet is not infrequent, as in other elderly people and is not necessarily due to cardiac failure (page 27) In advanced cases cardiac asthma (page 21) may be frequent and very distressing Anginal pain, occurring on exercise, and ultimately at rest, is the chief complaint of a number.

Recognition of High Blood Pressure

Accentuation of the 2nd sound —It is widely believed that change in the sound at the 2nd right cartilage means abnormality in the systemic, and at the left cartilage, in the pulmonary arteries, and accentuation of the 2nd sound has long been used, and more rigidly than is warranted, as evidence of raised pressure in the corresponding artery It is quite true that an accentuated second sound at the 2nd right cartilage is often heard in cases of high pressure, but such accentuation is not infrequent when blood pressure is unraised and, conversely, high pressure is not infrequent with the sound at or below its usual intensity It is to be remembered that it is not systolic pressure that is concerned in closing the valve and rendering it tense but aortic pressure at the very end of systole and that vibration of the valve is governed equally, if not actually to a greater extent, by the rate at which the pressure falls away in the ventricles at the end of systole Thus, while an accentuated 2nd sound may usefully draw attention to raised blood pressure it is invalid evidence of high arterial tension Similarly, the conclusion that pulmonary arterial pressure is raised cannot be based on an accentuated 2nd sound at the left cartilage

Pulse tension —The fingers should be trained to recognise a high-tension pulse, for a pressure meter can hardly be used as a routine in general practice The experienced will usually suspect high pressure, where it occurs, as soon as the radial pulse is felt To make more certain the index finger of the left hand should feel the pulse, while the index of the right presses proximally upon the artery to obliterate it The estimate of tension should never be attempted with the fingers of one hand only The amount of pressure that must

be exerted to obliterate forms the gauge A recurrent pulse will only occasionally make it essential to stop the artery low down as well

Sphygmomanometry—An accurate gauge of pressure at the top of the pulse beat (systolic pressure) in the brachial artery can be obtained by means of special instruments that are familiar There are many forms, and most are sound A few essentials must be named The gauge of pressure, if not a mercurial column, *should be checked against the latter from time to time* The outer surface of the cuff must be inextensible (leather or silk), the rubber bag, for use on arm only, must have a width not less than 12 cm and must itself be more than long enough to encircle the limb completely, otherwise over estimates of pressure become inevitable For very obese arms, and for the leg, larger cuffs should be used In taking systolic pressure, the cuff pressure at which the beats begin to reappear at the wrist is in general just as good a guide as is the return of the sounds over the artery below the cuff There are small errors and occasional fallacies with each method As pressure in the cuff gradually declines the sounds below the cuff become louder and later diminish in intensity These changes may happen once or twice during the decline The pressure at which the sounds begin to fall away abruptly and *finally* is the pressure usually taken as the index of pressure at the beginning of the pulse beat (diastolic pressure) It has been shown that normal readings of diastolic pressure, taken in this way, are sufficiently close to the true values, but abnormal very low readings are often less acceptable

In using systolic pressure fine distinctions of height are useless, *for the pressure varies much and frequently with pulse rate*, with emotion, exercise, and other causes A single reading has little value unless low, or unless exceptionally high, for previous exercise or present excitement will often lift the pressure to a high point in susceptible people (to 160 or 200 mm Hg) In adults repeated readings taken in circumstances of physical and mental rest, show normal pressures of 100 to 130 mm usually These are definitely normal values, but some would extend these limits in older subjects Values that are above 160 mm Hg definitely surpass normal limits Such readings are rightly regarded as less negligible in a young than in an elderly subject, because they are less usual The average blood pressure rises with age, this is not to say that rising blood pressure is normal, for many vigorous and long lived men retain low blood pressure, it is good *that an otherwise healthy but aged man should* show a pressure below the average for his years Resting pressures

of 180 mm and over are universally regarded as well beyond *normality at any age, though pressures often pass 200 and readings* up to 300 or even 350 mm are found

Often in cases of irregular heart action, and especially in auricular fibrillation, the beats pass the cuff at widely different pressures The cuff pressure that allows the occasional large beats to pass, while most are just obstructed, will form a useful, though it is not an accurate, gauge

Diastolic pressure lies nearer than does systolic to mean blood pressure and the latter expresses what the heart has to maintain, for this reason and because diastolic pressure reflects peripheral resistance more accurately than does systolic, theoretically it should contribute the more valuable index But diastolic pressure cannot be read with the same uniform accuracy as can systolic pressure, and because its range is smaller errors are more serious, therefore it is less used The normal resting readings are from 65 to 80 mm A pressure of 90 is usually and of 100 always abnormal In essential hypertension it is not uncommonly as high as 130 mm , occasionally it rises to 150 mm or more

Other things being equal, when the pulse slows, systolic pressure rises and diastolic falls

Accompanying and Final Manifestations

Many of the patients are heavily built or obese, high coloured, and becoming turgid in face when they stoop But there is no rule High pressure is also found in small people, and many lose flesh and become thin and anaemic in the later stages of the malady

Vascular manifestations —In addition to the signs of high blood pressure already detailed are those of general arterial disease (page 245) Disease of the arteries is invariable, often it is advanced

The rupture of small vessels is not uncommon, petechiae are easily induced and often occur spontaneously in the skin, purpura [illegible]

Apoplexy results when a capsular artery breaks, an accident that occurs almost exclusively in high pressure cases and is usually fatal

Thrombosis of the middle cerebral artery, an accident chiefly confined to high pressure cases, produces hemiplegia, with or with-

out aphasia Thrombosis of a coronary vessel too is often associated with hypertension

Transient hemiplegia, monoplegia, or aphasia is occasional Having a duration of a few hours or days, these disturbances are regarded as vascular, and are classed with convulsions, a rarer complication, as "encephalopathies"

Cardiac manifestations —*It is the rule to find signs of hypertrophy* of the heart in greater or less degree and the signs of left hypertrophy especially, *the emphysematous patient may not display this* imbalance The 2nd sound at the base is often accentuated or reduplicated at the impulse gallop rhythm (page 250) is a frequent sign Systolic murmurs are frequent at the base and less so at the apex The signs of aortic regurgitation or of mitral stenosis are found occasionally The rhythm of the heart is usually regular, alternation of the pulse, however, is frequent Of irregularities of rhythm, extrasystoles are common paroxysms of tachycardia are occasional Established auricular fibrillation occurs in a number of the cases The electrocardiogram usually displays the signs of left preponderance, sometimes it presents a grossly anomalous form (as in Fig 41, page 251) Angina pectoris or cardiac failure are common in the later stages.

event in
in which
ations of
pulmonary congestion are found In a few of these cases, *despite* great breathlessness *the systemic veins are undistended*, but it is the rule for the veins to be engorged in lesser or greater degree, *cyanosis is often inconspicuous* *Orthopnoea* nocturnal attacks of cardiac asthma (page 21) gallop rhythm and a subnormal tempera ture chart are the rule The asthmatic attacks are often severe Periodic breathing is present in many at this stage, though it may be no more inconspicuous waxing and waning of the breathing and must be watched for closely to be discovered, it is most emphatic during sleep The pulse, if regular, is rapid (100 to 120)

In other cases of cardiac failure with hypertension systemic venous congestion predominates, with its effects on kidney and tissue fluids, while breathlessness is not unusual and other mani festations of pulmonary engorgement are inconspicuous It is usual in hypertension for blood pressure to be maintained, when the heart is failing, until the last few days or weeks, sometimes the fall comes earlier, and it may then be reversed if *cardiac failure improves*

Thus hypertensives in the final phases present varying pictures which may be interpreted as resulting from failure of left or of right ventricle and different admixtures of the two (see types described on page 31)

Retinal manifestations —The arteries are often narrow and obscure the veins where they cross these Haemorrhages into retina or vitreous may happen Retinitis, manifested by small sharply defined white patches around disc or macula, is not uncommon

Renal manifestations —In most cases the urine is normal In some it is altered The quantity is increased to 1500 or 2000 c c , and the specific gravity is low Urine is often passed at night Albumin may be absent or occur in small quantity, with hyaline casts Renal efficiency is little impaired the urea concentration and clearance tests and blood urea being normal Oedema of renal type does not occur

In certain individuals, usually men under 50 years, a condition at first indistinguishable from essential hypertension develops a grave course, thought to be the result of obliterative disease of renal arterioles Severe morning or continuous headache and more ominously vomiting or grossly failing vision occur, albuminuric retinitis characterised by relatively large ill defined white patches along the vessels and macular region and obliteration of the margin with or without swelling of the disc is seen Blood pressure is exceptionally high the diastolic at 130 mm or more, polyuria is prominent, albumin granular casts, and blood cells appear in the [illegible]

[illegible] tensives with renal failure rather than by the objectionable name "malignant hypertension'

Inflammation —Hypertensive patients are unusually prone to [illegible]

[illegible] described do not necessarily or even usually remain isolated Cardiac, vascular, and renal events, and evidences of local inflammation, often become admixed

few survive them more than six or twelve months If as is frequent they are combined with periodic breathing, cardiac asthma, or alternation of the pulse, death is probable within a few weeks or months

The renal functions should be examined in all cases for signs of renal failure including albuminuric retinitis are also ominous whether they appear in combination with cardiac failure or alone

Treatment

Introductory —An important present day aspect of the care of high tension is to ensure that the patient's attitude to his condition is reasonable The public has been allowed to acquire incomplete

statements Patients in whom high pressure is found should rarely be informed of the fact, it is a technical detail that should not be allowed to concern them The habit of following blood pressure by frequent readings during treatment and of allowing patients access to such readings is strongly to be deprecated The patient becomes obsessed by blood pressure, and the manometer is regarded as the gauge of health and readings are watched closely and anxiously. Such use of the manometer is also a mark of ignorance, there is no virtue in the frequency of readings, and blood pressure is not in fact a chief, and not often a very important, guide to the progress of the malady Readings may be taken at intervals of months and in a manner to ensure that the measurement is regarded as but incidental It is often necessary that a patient should be made to understand that his activities must be limited, this can be done almost *always by a simple statement concerning advancing age and general* health, with at the most a reference to the circulation

It is better for patients in whom high blood pressure has been discovered, but who are without complaint or speak of no relevant symptoms, to have no active treatment, and, if the careful enquiry into the manner of living, which should always be conducted, reveals that they are *leading* reasonable *lives*, no change of *habits should* be made They should be under supervision only, and at intervals of a few months, it is unnecessary to them that they should be seeing doctors often

Mental quiet —Anxieties of mind are well known to raise blood

pressure and to maintain it when high Many patients suffering from high pressure are harassed people, full of business anxieties or of family difficulties and cares The relief of worry, if it can be accomplished, is material to effective treatment To the original troubles and to feelings of ill health, the dread of some more serious illness or disastrous accident must not be added On the contrary, these patients must be reassured and their minds placed so far as they can be at ease

Work and exercise —Hours of work that are obviously excessive should be shortened, strenuous forms of exercise or of manual work being forbidden Patients having few symptoms and no breathlessness or pain can continue in sedentary or light, or even in moderately light, manual work Indoor workers are to be encouraged to take exercise in the open air The amount of exercise is to be governed by the reaction of the patient, trials being made and the amounts increased gradually Walking, riding, and golf are suitable Exercise that produces distress of any kind at the time, or that induces subsequent headache, or fatigue that is more than brief, is unsuitable The effect of exercise upon subsequent sleep should be noted especially

Sleep —The pressure falls during sleep, and full hours should be ensured by suitable exercise, by abundant open air, and, if necessary, by hypnotics, of which bromides and drugs of the barbitone series are most suitable

Diet —Protein free or protein poor dietaries are not recommended A mixed diet such as the patient is accustomed to, reduced to simplicity and to a quantity adequate to maintain weight and no more (1500 to 2000 calories, according to the exercise taken), is best Some patients are stated to do better if salt is restricted It is doubtful if salt free diets are of value in these cases, and they are most unpalatable There is no objection to moderate amounts of alcohol in the form of spirits and light wines Beer or excessive quantities of fluid of any kind should not be drunk

Obesity should be treated by withdrawing first of all any excess of carbohydrates or fats from the ordinary diet, and later by a general reduction in the amount of food (to about 1500 calories value) Rigid dieting is not often well tolerated, it causes undue weakness, sudden reductions of weight should not be attempted Regular exercise helps to reduce weight, so does massage Turkish baths are not advised Thyroid may be given to stout women who develop high pressure at the menopause

Lowering blood pressure —The fear of cerebral haemorrhage and the frequency with which heart failure ultimately supervenes have suggested the use of remedies calculated to lower blood pressure Known substances and proprietary preparations have been and continue to be introduced in large numbers Nitrites, nitrates, acetyl choline, theobromine and other xanthine derivatives, benzyl benzoate, thyroid extract, liver extract are but a few of the more widely tried For each of these strong claims have been put forward, but none has come to be regarded as reliable or even as offering much or any prospect of benefit Perhaps the most striking reflection upon their value is the short period of popularity that each enjoys before it is displaced by a rival Even the long used remedy, potassium iodide, beneficial in some high pressure syphilitic cases, has fallen much in favour The amount by which pressure is lowered by these remedies is not often great, and the fall is either transient or restricted to the period of the drug's administration Many patients complain that they feel less fit under such treatment than without it

Recently, thiocyanate has been advocated The potassium or sodium salt is used in doses of 3 grains (0 2 g) thrice daily while the patient rests it is very potent in reducing blood pressure but it is not entirely without danger and should never be used unless closely controlled by estimates of thiocyanate in the blood the level in which should be carried to 5 and not surpassing 10 mg per 100 c c It effectively lowers pressure and patients usually speak well of it Nausea is frequent exfoliative dermatitis and increased weakness of the heart sometimes occur This and other drugs named may be tried in selected cases, but their administration should not be allowed to disorganise a patient s life, or to form the incentive to repeated blood pressure readings, or to divert attention from more natural remedies

Sympathectomy, consisting of bilateral removal of the splanchnic nerves and of the sympathetic chains from T9 to L1 has now been undertaken in a large number of cases It results in permanent lowering of blood pressure in at least half the cases so treated This drastic measure awaits the verdict of its final effects

A régime —The most successful general method of treating hypertension cases is by a careful regime, reducing physical and mental strains by imposing actual rest periods, short and frequent, or prolonged, by increasing hours of sleep, by shielding the patients from worry of any kind, by keeping the bowels lax with salines and the diet at the minimal quantity required to prevent undue loss of

weight, by forbidding tobacco altogether or in anything but most moderate quantities Such a régime, associated with intervals of light open air exercise and quiet relaxations or amusements, can be carried out at home, but often more readily at some soothing country spot or quiet seaside place It is often effectively carried out at a spa, where it will usually be combined with some particular local remedy, such as warm baths, saline or gaseous, that produce mild cutaneous vasodilatation, or mineral water drinks, such will usually come to claim most of the credit for any benefit derived from the régime as a whole

Headache is often relieved by the use of high pillows at night Warm baths, aspirin or phenacetin (10 grains or 0 6 g), are each and often effective A very drastic dietary confined to vegetable soup and fruit juice for 5 or 6 days has been advocated It is sometimes very efficacious Venesection, and lumbar puncture may be used in persistent severe headache

Angina pectoris —In angina of high pressure the usual remedies are employed

Cardiac failure, and asthma —The treatment of cardiac failure has been dealt with earlier There are a few supplementary notes to add It is important that treatment by rest in bed should be used early in the cardiac failure of hypertension, namely, when breath lessness occurs during brisk walking exercise and before signs of congestion appear Several months of complete rest will usually relieve for a much longer period, it should be followed by long rest *periods each day, and especially after meals, for the rest of life*

Anasarca is not often massive but, if it should be, strictest precautions should be taken when draining the legs, sepsis is much more likely to appear than in the younger cases of rheumatic heart disease The usual diuretics may be used, but mercurials only if there is no renal failure

A period of complete rest is also indicated when breathlessness without signs of congestion, or attacks of nocturnal breathlessness, appear Some of these patients react well to full digitalisation. Treatment of the attacks themselves has been given on page 42.

CHAPTER XXV

ARTERIOSCLEROSIS SENILE HEART MYOCARDIUM

Arteriosclerosis

After the age of thirty the arteries of the body are rarely quite normal, and they change increasingly though varyingly with advancing years Arteriosclerosis that comes early and progresses quickly runs in families The disease is well known to be associated with high blood pressure, but often occurs apart from this, it is stated to be more prevalent in those consuming much alcohol and in those engaged in heavy manual work Its cause, however, is unknown

PATHOLOGICAL ANATOMY

The earliest signs of arteriosclerosis are to be found usually in the aorta, where small yellow flecks are visible in the intima In more advanced cases the aorta is studded with small and larger nodular masses of the same fatty or gelatinous substance The deposits occur early around the mouths of outgoing vessels Ultimately the whole normal intima is concealed and rendered irregular by the deposits, in which rough calcareous plaques are laid down and by denudation of the surface The thickening of the intima is generally regarded as compensatory to atrophic changes in the medial coat, the adventitial coat becomes thickened and sclerotic In very advanced cases the aorta may be converted into a rigid and brittle tube, or it may become aneurysmal Similar changes can occur in all the arteries of the body, they are usually affected in the order of their size When affecting smaller arteries the disease may fall with especial heaviness upon a given group of vessels like those of brain, kidney, heart, or limb Damage to the tissues in the corresponding territory occurs when the flow of blood through the affected vessel declines This happens in one of several ways Thrombosis in a patent vessel may yield acute and massive necrosis, which may or may not become converted ulti

mately into a local scar When the wall of a smaller vessel becomes thickened, the lumen often narrows materially so that little blood or, when the tone of its wall increases, no blood can pass and with inadequate collateral channels, the nutrition of the tissues in its territory will suffer Thrombosis completing a closure already far advanced may happen quietly Consecutive changes may be brought about in the muscle of the heart in one or other of these ways, and consists of fatty degeneration or replacement fibrosis

RECOGNITION OF ARTERIAL DISEASE

Advanced arteriosclerosis of the aortic wall leads to loss of elas ticity and to reduced resistance of its walls the aorta elongates, be comes a little sinuous, and dilates (Figs 39 and 40) These changes

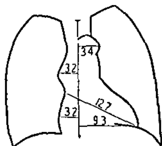


FIG 39 —Orthodiagram Man aged 59 Advanced general arteriosclerosis angina pectoris gout Systolic blood pressure 165 mm No valve disease Wassermann reaction negative The aorta is very tortuous its probable course is marked by dotted lines

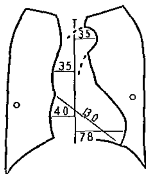


FIG 40 —Orthodiagram Man aged 63 weight 108 lb Advanced general arteriosclerosis systolic blood pressure 310 mm No valve disease The ascending aorta is elongated and unusually curved

can occur independently of, but are exaggerated by, high blood pressure Occasionally, with nicely adjusted exposures calcareous deposits in the aorta are displayed radiographically, as they can be more frequently in the vessels of the limbs

At the bedside, arterial disease is recognised by noting prominence of the vessels in their courses and by their tortuosity In the normal arm of the young adult no pulse is visible but in arterial disease pulsation appears over the course of the vessels, at first where the vessels normally lie most superficially, as at the wrist and before the elbow joint, ultimately throughout the whole length of the upper arm, and even on occasion throughout the forearm The pulsa

tion is naturally more conspicuous in cases in which the pulse is large, and it is also more conspicuous when the arm is flexed, for the slack in a vessel that has lost its elasticity is not taken up when its course is shortened, and so the artery is forced to become sinuous When the disease is more advanced, the vessel is actually elongated and its course is then tortuous when the arm is extended Some times tortuosity is so great that the brachial artery is bent until its course is reversed for a short distance, and in very rare cases may actually make a complete loop in the arm Tortuosity of the super ficial temporal vessel is of less value because these vessels natur ally follow a wavy course and, when dilated their very superficial position renders them unusually prominent

At the wrist, thickening of the radial artery may often be felt A length of vessel should be emptied of blood by occluding it above, wiping out the blood and occluding it also below If the stretch of empty vessel between can then be felt and rolled under the finger, or if its surface is irregular, it is diseased A normal artery when full and tense can often be rolled under the finger in this way

Strictly speaking disease is only recognised in the artery in which it is observed, but because arteriosclerosis is almost always wide spread, conspicuous changes throughout the vessels of the arm may be used as sufficiently convincing evidence of widespread disease It cannot be used to diagnose appreciable disease of particular vessels, coronary or cerebral, though it does indicate that the case is of a type in which such disease may be expected to exist, and it helps to interpret corresponding groups of symptoms in cases of doubt Irregularity of the retinal vessels provides a further hint

CONSEQUENCES OF ARTERIAL DISEASE

Although essential hypertension and arteriosclerosis are separate pathological conditions, they are very often associated That is so because they are both common maladies of the same age period and because, when there is a tendency towards arterial change, its development seems to be aided by high pressure, it is a mistake to think that diseased arteries cause high pressure

Essential hypertension is so frequent and so important clinically, while at the same time sufficiently defined, that it is important to give it the emphasis of separate portrayal By so doing, many of the phenomena associated with, and originally regarded as, simple manifestations of arterial disease, become included under essential

hypertension Some of these, for example cardiac hypertrophy, are properly regarded as pertaining to high pressure and not to arteriosclerosis, which, unless extreme, throws little burden upon the heart Others are to be regarded as pertaining to both, thus angina pectoris in part results from an increased burden, and in part from the disease of coronary vessel, and cerebral haemorrhage, while occurring almost exclusively in high-pressure cases, would not happen unless the unusually strained vessels were diseased Lastly, there are phenomena that pertain to arterial disease itself, for example thrombotic accidents Very many of the manifestations described as accompanying essential hypertension might, with equal reason, have found their place in this chapter To divide strictly and accurately is impossible and the attempt would have separated phenomena that are usually found together The present brief account is intended in part to correct any false impressions that might otherwise arise, and in part to supplement

Apart from rupture of the diseased vessel wall, an accident sufficiently noticed in the last chapter, the material consequences of arterial disease are nutritional The special symptomatology of arteriosclerosis is to be found in the abrupt failure or decline of blood supply to various organs, such as the brain, the kidneys, the pancreas, the limbs, and the heart The more important cerebral manifestations have been named in the last chapter Disturbances of cerebral nutrition are apt to sum themselves into a state of senile dementia, accompanied by symptoms like those of Parkinson's disease The deeply scarred arteriosclerotic, contrasting with the granular arteriolosclerotic, kidney has no great clinical significance In the arteries of the pancreas disease may give rise to diabetes, in the vessels of the limb to distal gangrene or, when arteries to the muscles of the calf are involved, to intermittent claudication Acute closure of the coronary vessels leads to the syndrome fully considered in Chapter VII gradual closure is responsible for angina of effort, and by decreasing the musculature and robbing it of strength, leads up ultimately to failure with congestion, clinical states already considered at length

A frequent association of arteriosclerosis and the degenerative processes of advancing years is auricular fibrillation Its development may determine failure with congestion, but more often the change passes unnoticed, the rate of ventricular response being slow in the elderly Alternation of the heart beat is often seen

Heart Disease in the Aged

PATHOLOGICAL ANATOMY

Coronary arterial disease is the chief feature The senile heart may be enlarged apart from high blood pressure not infrequently it is normal in size or may even be a little reduced There is often a diffuse increase of fibrous tissue in it apart from such larger and denser areas that have arisen obviously from vascular thromboses The muscle is often pale, and special stains reveal diffuse fatty degeneration It may be brown in colour and show pigmentary degeneration

An ascending degeneration of the aortic cusps, of which the mild forms are common in adults is distinct pathologically from arterio sclerosis, which usually accompanies it It begins in the substance of the cusp near its base and passes on to thickening that spreads up the cusp Then large calcareous deposits occur at the bases of the cusps and project into the sinuses of Valsalva The craggy masses cause obstruction This degeneration is often responsible for aortic stenosis as this occurs in elderly people Similar degenerative change sometimes happens in the aortic leaf of the mitral valve and calcareous deposits are laid down at its base, and in the adjoining part of the ventricular septum, in the last situation occasionally breaking through the auriculo ventricular bundle

CLINICAL FEATURES

In elderly or in old people the heart presents many different clinical pictures There are instances of long standing rheumatic disease of the aortic and mitral valves There are long surviving, or late developing cases of syphilitic aortitis But the degenerative lesions named earlier dominate in the cardiac disease of old age and produce the acute phenomena of coronary thrombosis or spasmodic angina, and failure with congestion, predominantly pulmonary, often super venes upon one or other of these, or occurs without these prelimin aries, as the amount of sound muscle in the heart dwindles Here too must be mentioned again craggy aortic stenosis and the occasional case of heart block which may result from similar calcareous lesion or from fibrosis in the ventricular septum The development of angina will be expedited, as will failure with congestion in other cases, by increasing arterial pressures, which are so frequent with advancing years Terminal bronchopneumonia is frequent These various types and complications, occurring singly or in combination, have been described already

There remains for brief notice the heart that fails in old age without displaying any of the major antecedent troubles and in a less conspicuous way As age advances a general lowering of vitality occurs and nutrition begins to fail and the body wastes A systolic apical murmur appearing for the first time may have value as an early guide to a dilating heart The bodily reserves have usually begun to decline long since and among these is cardiac reserve displaying itself by breathlessness or pain on effort In the decline there is often seemingly a slow race between cardiac capacity and the powers of the body generally The heart is losing its reserve but the man is becoming enfeebled owing to local arterial trouble or general weakness his limbs serve him less well, his will or inclination to carry out his old pursuits declines he requires and takes less food and thus as the heart weakens so simul

sometimes lengthening it remarkably though always precariously The balance is unstable since the reserves are gone and is quickly broken by any unusual event such as an enforced period of activity or an infectious disease such as bronchitis or pneumonia In old men failure when it comes terminates life quickly and often with relatively few signs The aged pass out unobtrusively after brief illness or without warning while sitting in their chairs or sleeping in their beds

The Myocardium

In this and previous chapters much has been said of cardiac failure and of anginal pain and very little directly about the muscle of the heart This is as convenient a place as any to consider the relation of diagnosis to the myocardium There are some physical signs as yet undescribed which are relevant from this standpoint These will be discussed briefly with comments upon their values

SOME PHYSICAL SIGNS OF MYOCARDIAL INVOLVEMENT

Pulse strength —The size of the pulse rarely has much value in gauging the state of the cardiac muscle it is largely controlled by the local state of the artery and by the rate at which the heart is beating Thus the pulse is normally much smaller when the hands are cold than when they are warm and it is usually very small in

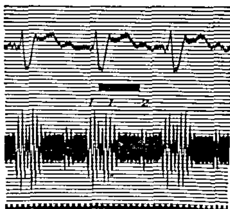
paroxysmal tachycardia Decreased pulse tension (low blood pressure) is not much more significant It is true it may result from deficient output of the heart at each beat, consequent upon weakness of the cardiac muscle, but it may result also from deficient return of blood to the heart, as in haemorrhage, and as in profound loss of vasomotor tone Central and peripheral factors are often combined and notably in the acute infectious fevers The pulse weakens to imperceptibility in the last stages of many diseases including cardiac failure, but by that time the state of the patient is usually apparent at a glance It is to be remarked that patients frequently present advanced signs of cardiac failure and high blood pressure simultaneously

Heart sound intensity —The intensity of the heart sounds, and especially of the 1st sound, has long been thought to throw light upon the strength of the heart It is true that decrease in the intensity of the 1st sound often occurs when the circulation is failing rapidly as in the acute infections, a failure that is in part cardiac and in part vasomotor It is also true that it occurs in terminal cardiac failure but at a time when there is a redundancy of signs Ordinarily, the intensity of the sound varies greatly according to the amount of covering of the heart and chest wall, according to the way in which the heart is acting, and with less easily determined factors Emphysema and obesity both greatly diminish the intensity of the sounds Simple but very rapid action, as in paroxysmal tachycardia weakens the 1st sound relative to the 2nd, and produces sounds having much the spacing and intensity of the foetal heart sounds It is not questioned that weakening of the heart beat results in lowered intensity of the sounds, it is merely pointed out that other factors are involved Actual experience shows that in gauging the essential soundness of the heart, in patients who are up and about the intensity of the sounds helps very little Similarly, signs based upon change in the character of individual sounds, though of some theoretical interest, are insufficiently precise, and lack enough constancy in given circumstances, to be of any great practical value

Gallop rhythm —This is constituted by three distinct heart sounds, usually of almost equal loudness Usually the heart rate in these cases is a little rapid (90 or 100), the gallop is best heard at the impulse though often over a wide area The extra sound is found in graphic records (Fig 41) to lie usually in presystole or in mid diastole and is often clearly related to the contraction of the auricle Gallop rhythm is found especially in hypertension cases in which

the heart is failing, or in the heart failure of elderly subjects There is as a rule no valve disease The sign is often associated with prolongation of the auriculo ventricular conduction interval, or with bundle branch block The cardiac impulse is often double Gallop rhythm may help to determine the cardiac origin of breathlessness or pain, by demonstrating the heart to be abnormal, for only patients with abnormal hearts present this sign, and they are usually serious cases There is no constant underlying lesion, though hypertrophy of the auricle is common

Wide reduplication of the 2nd sound, which also gives a sort of gallop rhythm, occurs especially in cases of mitral stenosis with slower heart action The extra sound, in the early part of a long diastole, is unrelated to auricular systole, as it may occur when the auricles are fibrillating, it is probably due to an unusual intensification of the normal 3rd heart sound, and tends to be confined to the region of the impulse





"T" wave inverted — In young and healthy adults T is always upright in an electrocardiogram taken from the right arm and left leg An inverted T (Fig 23, page 124) is abnormal, but occurs in a number of conditions Temporarily it is seen under heavy dosage with digitalis or after drinking quantities of ice-cold water As a persistent condition it is common in elderly heart patients, and most frequent in those in whom there are abundant other signs of cardiac weakness It frequently appears a week or more after coronary thrombosis (Fig 6, page 57), and often persists Although the death rate among those displaying it constantly is heavy, it has been known to persist for twenty years in a patient whose health remained unchanged during that period It should not be used for prognostic purposes

Bundle branch block —Large abnormal electrocardiograms in

which the initial phases are very wide and the chief and final deflections are in opposite directions (Fig 42), are caused by interference with one or other branch of the auriculo ventricular bundle The pathology of the condition and its clinical associations are similar to those already described under heart block, with which it is often combined, though bundle branch block is most frequent in cases of aortic disease and coronary disease As a temporary condition it indicates active mischief in the heart and may be the only clear indication of this As a persistent condition it is again significant of a myocardial affection As in the case of heart block, it is usually to be regarded as indicating changes that are not confined to the special tract, but more widely diffused It is almost always accompanied by abundant cardiac symptoms and signs pointing to serious cardiac involvement, such as those of cardiac failure, anginal pain, periodic breathing gallop rhythm, pulsus alternans and attacks of nocturnal breathlessness and most of the patients who exhibit it are dead within two years But some, and especially those in which there are not the ominous associations survive for many years and so the prognostic significance of the sign by itself is indecisive Its chief value is that in cases of obscure breathlessness or pain it may indicate the heart as the seat of mischief

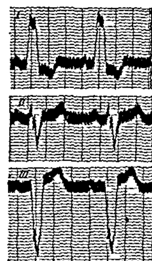


FIG 42 —Electrocardiogram from leads *I II* and *III* illustrating the common type of bundle branch block. The main deflections in leads *I* and *III* are in opposite directions The initial and final deflections in each of these leads is opposite in direction The initial deflections in each lead are conspicuous in amplitude and unduly prolonged Time in ⅕ sec

Pulsus alternans —The pulse beats are rhythmic but alternate in height (Fig 43) Its cause is thought to be failure of a proportion of the fibres of the ventricle to contract at alternate beats of the heart Alternation often occurs when the heart is beating very rapidly, it is usual in auricular flutter, and frequent in paroxysmal tachycardia It occurs also when the heart is beating slowly, and is then most frequently associated with hypertension, cardiac failure, angina, coronary thrombosis, or syphilitic aortitis Its heaviest incidence is between the years fifty and seventy Whenever it occurs there is reason to believe that the left heart, or a substantial part of it, is struggling

to perform work of which it is scarcely capable If the heart is disposed to alternate, added strain, such as high blood pressure or rapid beating, will determine it An extrasystole will often disclose alternation for a few cycles (Fig 44)

Although of common occurrence, it cannot be recognised by feeling the pulse or cardiac impulse, but must usually be shown



Fig 43 —Simultaneous electrocardiographic and radial pulse curves from a case showing alternation The radial curve consists of alternate large and small pulse beats as usual the length of the large cycle is rather greater than that of the small one In this instance the electrocardiogram shows no alternation but perfectly regular and seemingly normal heart cycles Time 1/5 sec

sphygmographically Not infrequently, when persistent, it can be detected by means of the blood pressure apparatus, alternate beats forcing their way through the cuff at pressures 5 or 10 mm Hg higher than the remainder

Occurring at high rates of beating it has relatively little significance If persistent at slow rates of beating it is a grave sign Few patients displaying it survive for one, and very few for two,

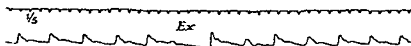


Fig 44 —A radial pulse curve showing alternation in its second half The alternation has been provoked by the extrasystole (Ex) which stands in the middle of the curve Time 1/5 sec

years It is usually accompanied by abundant other evidence of grave heart affection, signs of failure, angina, attacks of nocturnal dyspnoea, periodic breathing, gallop rhythm, or bundle branch block Though never the sole sign of disease, persistent alternation is occasionally found in the absence of the named phenomena and then still retains its ominous significance There is no lesion of the muscle with which this curious and important disorder of beating is known to be correlated.

MYOCARDITIS FATTY AND FIBROID HEART

Three morbid conditions of the muscle alone call for brief consideration here They are "myocarditis", "fatty heart", and "fibroid heart"

Myocarditis —The only form of inflammation of the heart musculature that is at all common is rheumatic in origin, and it takes an acute, or more often subacute, form Occasionally inflammation occurs in other acute and fatal infections, diffusely in diphtheria, locally in pyaemia The term myocarditis has often been allowed most loosely to comprise all altered states of the muscle, real or supposed It has been employed improperly as an equivalent of myocardial weakness Actually the use of the term myocarditis is scarcely to be justified clinically except in relation to rheumatism, and here the term "carditis" is sounder The description of myocarditis acute or chronic as a separate disease with its special symptoms and signs needlessly complicates a general description of cardiac disease

Fatty heart should not be diagnosed If a patient has cardiac pain and is obese, *it is easy to guess that around the heart an unusual* amount of fat has accumulated The guess however, would be equally correct if there were no pain A diagnosis of fatty heart in such circumstances has no more value than has that of "obesity", *and thus it is objectionable because it pretends to be more than it is* If we mean by fatty heart a fatty degeneration of the muscle, then this cannot be diagnosed with any pretence to accuracy, unless perhaps in acute poisoning by infective disease or phosphorus, or in anaemia, and in these the desire to make the diagnosis is infrequent In severe diphtheria the cardiac muscle may be so degenerate with fatty particles that it is throughout of a pure yellow colour, yet the heart continues beating Because diffuse fatty degeneration occurs in the senile heart, or in coronary arterial disease, that is no reason for regarding fat in the heart as a cause of sudden death Equal change is found in the heart of pernicious anaemia, and here sudden death is most unusual If a man dies suddenly in the street, a court is required to find the reason, and if nothing else is forthcoming, "fatty heart" by usage proves convenient and acceptable The enquiry may not rest upon the answer "Death from natural causes" unless an anatomical basis for death is found There is evidence that the ventricles of elderly people, and especially those presenting coronary disease, are prone to fibrillate This fatal fibrillation is an

event in living muscle, it leaves no message written upon the dead heart The conception that correlates fatty heart and sudden death may be expedient in forensic practice, but, while fatty heart is usually unrecognisable in the living and its prognostic significance is uncertain, the conception can find no place in clinical diagnosis

Fibroid heart—Fibrosis in the heart is found in one of two chief circumstances Firstly, it occurs very diffusely, as an end product of rheumatic inflammation, it is then mainly an interstitial though in part a replacement fibrosis Secondly, it occurs in the senile heart, either as an after effect of vascular thrombosis, and representing areas of healed infarction, or more diffusely and more commonly as a consequence of diminished nutrition in narrowing of the coronary arteries and arterioles, these two forms of fibrosis are often combined That fibrous tissue is present in excess in the ventricle may be inferred from a history of rheumatic carditis or of coronary thrombosis but its extent cannot be ascertained The patient's out look depends not on the fibrosis, but upon the state of the remaining muscle For these reasons fibrosis of the heart should not be diagnosed, the patient's condition being summed up in terms of its functional ability

MYOCARDIAL WEAKNESS

It will be gathered from what has gone before that an attempt to correlate physical signs with specific states of the cardiac muscle offers prospect of infrequent success Inflammation of the myocardium can be diagnosed safely in subacute rheumatism when the heart is clearly involved, but the intensity of the morbid change, the numbers of submiliary nodules present, cannot be gauged The weakness of the muscle correlates with more subtle processes Fatty degeneration of the muscle in chronic heart disease is of unproved significance and cannot be diagnosed accurately Fibrosis of the heart can only be diagnosed satisfactorily when the fibrous tissue has followed locally a coronary thrombosis Myocarditis, fatty, and fibroid degeneration are the only gross changes that are at all common in the heart Heart block or bundle branch block may indicate focal lesions in the conducting tissue, and may allow us

the basis on which to diagnose myocardial lesions is slender Moreover, many patients dying of cardiac failure show little change of any of these kinds

An outstanding fact of cardiac pathology is that we are unable to demonstrate after death, the changes in cardiac muscle that have led to its failure, minor changes are found in failure, but they are inconstant in kind, occurrence, and degree We are rarely able to predict before death the specific changes that will ultimately be found in the ventricle after death, and never the extent of the change It is sounder in practice not to make the attempt to diagnose in terms of anatomy, but in the more certain terms of function Angina pectoris and cardiac failure are clinical states, which may be regarded as the chief products of defective nutrition or of weakness of the myocardium It is in these terms that diagnosis must usually be made, because they convey with sufficient precision states that are recognisable and measurably significant When we merely substitute for the functional diagnosis such terms as coronary disease, myocardial disease or whatever is supposed to be the underlying basis of the disturbance we introduce assumption and, while gaining nothing substantial, sometimes depart gravely from truth Angina pectoris and cardiac failure never can find equivalents in the terms of structure, for each depends not only upon something in the heart but upon something that the heart is called upon to struggle against that lies outside itself

The desired end point the diagnosis of heart failure, in one or other of its types and degrees, or of angina pectoris, is often aided by the proper use of certain of the physical signs described earlier in this chapter A patient has breathlessness or pain, but in neither instance is the origin of the symptom manifest, the diagnosis will often be brought to the point of justification, or actually be rendered certain by the finding of a persistent phenomenon such as auricular fibrillation, heart block, bundle branch block, gallop rhythm, or, a little less emphatically, by inversion of T For each of these phenomena points to definite abnormality of an organ from which the symptom under consideration often is derived Alternation of the pulse is of decisive value, for it not only shows the heart to be abnormal, but actually presents clear evidence that the heart, or a substantial part of it, is labouring under difficulty.

CHAPTER XXVI

PULMONARY HYPERTENSION COR PULMONALE FAILURE IN PULMONARY DISEASE RIGHT SIDED FAILURE

It should be understood that in speaking of pulmonary hypertension we are speaking largely hypothetically we do not measure pulmonary pressure but judge it to be raised from theoretical consideration or from indirect physical signs

Pulmonary hypertension is regarded as happening most frequently as part of pulmonary congestion and is then a consequence of an obstructed outflow from lungs to left heart as described in Chapter III and under mitral stenosis (page 135) It is therefore a common association of heart failure

But attention is now to be directed particularly to states important but sometimes obscure in which it is considered probable that pulmonary arterial pressure is raised by an obstruction in the lungs themselves

Acute Cor Pulmonale

The condition to which this name has been given usually arises out of massive embolism of the pulmonary tree from clots dislodged from the systemic veins and especially those of the leg in elderly surgical cases or from the pelvis in obstetric cases It is to be remembered that the arteries of the lungs are so numerous and wide that a very large proportion of them more than all those running to one lung can be blocked without appreciably affecting pulmonary arterial pressure Examples of pulmonary embolism arising out of heart disease (Chapter III) are rarely of this order A single embolus completely blocking right and left pulmonary arteries brings almost immediate death multiple emboli blocking a large proportion of the arterial channels produce a very grave condition one that is not immediately fatal and from which recovery may occur

SYMPTOMS AND SIGNS

The chief symptoms are a sense of intense pressure or pain over the sternal region suffocation breathlessness and collapse

resemble the deepest cyanosis of congenital heart disease Even when venous pressure is relatively low, cyanosis and breathlessness may be deep and accompanied by polycythaemia and even finger-clubbing When fits of coughing come, the cervical veins swell greatly and the patient's skin and lips become deeply suffused and cyanosis reaches an extreme point

Special signs pointing to overstrain of the pulmonary circulation, are the loud and abrupt, sometimes duplicate 2nd sound at the pulmonary cartilage, and evidence from percussion and X ray that the pulmonary artery is enlarged

Emphysema —In this the chest will be observed fixed in the extreme inspiratory position, it is short, deep, and presents the familiar wide intercostal angle The chest wall is rigid and its movement small expiration being particularly ineffective Evidence of the expanded lungs is found in the unusually hyper resonant note, and in the extension of lung resonance over the heart and beyond its usual limits over the lower ribs The recession of the heart from the chest wall obscures its impulse and its margins abolishing superficial cardiac dulness, it is impossible to determine the heart's size with accuracy, except by X ray examination The sounds of the heart become distant, and are obscured by the coincident noises of bronchitis.

Silicosis —This condition is frequently accompanied by compensatory emphysema, which tends, as in the last instance to obscure cardiac signs The diagnosis will depend on the history, with its relevant occupation, and always for the extent of disease upon the X ray examination

PROGNOSIS AND TREATMENT

In emphysema the immediate prognosis of cardiac failure depends upon the course of the bronchitis If this has been chronic, the outlook is unpropitious If an acute or subacute attack is relieved the signs of cardiac failure clear away also, actually recovery usually occurs in these attacks, though it is gradual and may be spread over several or many weeks The patient may or may not be left measurably the worse after the attack is over Extensive infarction of the lungs sometimes kills these patients

When patients with emphysema or silicosis begin to show definite signs of cardiac failure, whether in attacks or in exacerbations of bronchitis or otherwise, they are entering upon the last stages of their malady Life is not usually prolonged for more than one or

two, exceptionally for a few, years This prognosis conforms to wha is usually found in failure with congestion when this develops ir middle aged or elderly men

The treatment of the cardiac failure is carried out on lines simila to those already laid down Rest and efficient nursing are essential Auricular fibrillation when present is treated appropriately Vene section is useful when the veins are much engorged, depth of cyan osis is not the guide Efforts should be directed to relieve the strair of coughing Ammonium carbonate or chloride (5 grain or 0 4 g doses four times daily) are given when the sputum is scanty and viscid potassium iodide may be used in similar doses for the same purpose Ipecacuanha may be employed if it causes no nausea Relief may be secured by saturating the air which the patient breathes with moisture by the use of the steam kettle The patient should endeavour to avoid strain in coughing and may be helped to do so [illegible]

should avoid dusty atmospheres, public buildings and crowded rooms

CHAPTER XXVII

THYROTOXIC STATE

INTRODUCTORY

CHIEF manifestations of the thyrotoxic state, and the most diagnostic clinical signs of the disease are to be found in the thyroid gland itself and in the eyes The condition occurs chiefly in women between the ages of twenty five and forty five It is enough here to name the firm enlargement, either uniform or nodular, and the vascularity of the gland, together with the exposed, staring eyeballs Associated with these most characteristic phenomena are others There is change in the nervous system, represented by an alertness, restlessness, quickness of movement, emotionalism, and tremor of the hands Sweating is in excess there is sometimes a diffuse pigmentation of the skin Metabolism is increased, and with this may be linked wasting, weakness and occasional glycosuria

Lastly, in all but exceptional cases, there are disturbances of circulation that here claim particular attention they may be crucial in leading to diagnosis The circulatory system is found to be in a state of increased inactivity, which is often remarkable and always important The development of circulatory failure sometimes jeopardises life, and it leads too often to the postponement of surgical treatment.

CARDIOVASCULAR MANIFESTATIONS

Pathological anatomy —In patients dying of cardiac failure the heart is found to be dilated and not infrequently hypertrophied, though not greatly No distinctive histological change has been found in the muscle, which is usually little altered

Vasodilatation —Cutaneous vasodilatation is the rule, and evidence of it is often conspicuous The two most valuable signs are increased temperature of the skin and capillary pulsation It is increased temperature of exposed parts such as face and hands that

must be observed, the face feels hot and the hands keep warm or hot when outside the bed clothes in cool or cold rooms The patient feels hot and prefers cool room temperatures to warm The face is often flushed, sometimes deeply, and this flush extends to the neck and covers the body, though less conspicuously It is a mistake to regard this depth of colour as important, freshness of tint, and associated warmth and capillary pulsation, are of more consequence Not infrequently the depth of colour is natural or there may even be pallor Capillary pulsation is always to be seen in the face when there is appreciable vasodilatation, though experience is required to detect it *As a spontaneous event it is plainest in the cheeks and* forehead, and should be looked for with keen attention from a little distance The skin should not be rubbed but a glass slide may be pressed on cheek, lobe of ear, and lip if the pulsation cannot otherwise be detected in face or pads of fingers

Pulse —When regular the pulse is rapid the rate being often as high as 100 to 120 while the subject rests and rising higher with exercise or excitement it is high in sleep The stroke is full and abrupt Systolic blood pressure is in general raised, ranging around 140 or 150 mm Hg, diastolic readings show less rise Water hammer pulse may occur

Precordial signs —The patients are usually thin and the heart beat is augmented Therefore a diffuse impulse is customary, it almost slaps the examining hand and not infrequently imparts the feeling of a slight thrill The maximal impulse is often displaced outward an inch or a little more in the 5th space The character of the impulse is apt to convey the impression of a larger heart than is there Abrupt and accentuated heart sounds are frequent, a systolic murmur is common over 2nd and 3rd left interspaces and, when the impulse is displaced outward over this also A cardiorespiratory murmur in the left axilla is usual, when the heart is overacting

Auricular fibrillation is present, either in paroxysmal form or as a continuous affair in one fourth to one third of the cases as these come for treatment The ultimate reason for this frequent and important accompaniment is unknown though it is recognised to be

ventricular response to the fibrillating auricle tends in these cases to

be high When fibrillation comes, it increases the degree of cardiac failure or introduces it Quite occasionally flutter of paroxysmal tachycardia takes its place

Cardiac failure—Breathlessness on effort, the symptom of failure is the rule in these patients This failure is to be ascribed to overaction of the heart in the presence of thyroid intoxication The energy expenditure of the heart is increased in part because its rate of beating is high, and in part because its output of blood is raised, and this output takes place against a systemic pressure that is often above the normal Systemic venous congestion super venes in some, being chiefly confined to those presenting auricular fibrillation In identifying venous congestion, stress should not be laid upon engorgement of the cervical veins in patients who present signs of increased vascularity of the thyroid gland For, when the gland has a thrill over it, many large veins can often be seen returning blood from its region in unusual quantity, and this tends to overfill the cervical veins as a whole Here enlargement of the liver is a more valuable sign than swelling of the cervical veins

Anginal pain, at first with effort and later at rest, occurs in some of the more elderly patients, whose auricles may or may not be fibrillating It may be severe, and the attacks repeated by day and night It probably occurs only in those predisposed to it apart from the thyroid disease

Diagnosis

The diagnosis of the thyrotoxic state presents no difficulty when exophthalmos, a thyroid tumour, and overacting heart are found together It is the latent case, in which ocular signs are absent and the thyroid is but little enlarged, that presents difficulty, and in such the suggestion that the thyroid gland is at fault comes first from the circulation All cases of paroxysmal auricular fibrillation should be considered from the standpoint of thyroidism, so should cases of persistent but recent fibrillation The question may also be raised advantageously in cases of continued fibrillation that are unexpectedly resistant to treatment If distinct symptoms of cutaneous vasodilatation as previously detailed are present in such patients, the diagnosis is probable Unusual coldness of the hands is strongly against the diagnosis If similar symptoms of vasodilatation are associated with a long continued and unexplained simple tachycardia, the thyroid is to be suspected The occurrence of severe thyroid intoxication in middle aged women with little thyroid en

largement is to be emphasised In these types hints may be obtained from nervousness of the subject from slight diffuse pigmentation from occasional but unexplained diarrhoea from loss of weight and from signs revealed by close scrutiny of the thyroid An estimate of basal metabolic rate will usually decide A rate raised more than 25 per cent above the normal value is considered in the absence of fever or other recognised cause to be strongly suggestive values raised 40 or 50 per cent or more are not infrequent A normal basal rate excludes hyperthyroidism a matter of occasional importance in differentiating cases presenting severe effort syndrome (page 168)

Treatment and Prognosis

Whether it is looked at from the standpoint of the heart or not the first essential in treatment is complete rest During this initial period the patient is becoming accustomed to the surroundings and the pulse rate will often fall a good deal without further treatment Active treatment follows one of three lines alone or in combination

Iodine is given in doses of 5 minims (0 3 c c) of a 5 per cent solution in 7½ per cent of potassium iodide taken well diluted in water thrice daily it lowers metabolism temporarily the pulse rate falling to 90 80 or even 70 per minute and symptoms subsiding A maximal effect is usually obtained in 10 to 15 days but is not maintained though some mild cases seem permanently benefited from one or two courses

Subtotal thyroidectomy —Since iodine fails as a continuous remedy it is used chiefly in preparation for surgical intervention If the maximal effect is passed on the first course it should be reached in a second course after a two weeks interval and the operation then done This preparatory treatment to subtotal thyroidectomy greatly decreases the risk of post operative thyroid intoxication and from the same standpoint iodine is often continued for a week after operation Time has established such treatment on a firm basis Under this régime and in the hands of a skilful and experienced surgeon the mortality from the operation is 1 per cent or less the beneficial effects are usually conspicuous and long lasting In some cases removal of a second portion of the gland becomes advisable owing to recurrence of symptoms

Patients who have already had paroxysms of auricular fibrillation often develop an attack during the first two days following operation Such patients may be given digitalis before operation otherwise they should be brought under the influence of the drug rapidly (page 99)

as soon as fibrillation appears, if the patients are sick, the digitalis may be given by rectum Quinidine may be used as a prophylactic over the operation period

In patients presenting persistent auricular fibrillation, digitalis should be used at once to lower excessive ventricular rate, and should be continued until signs of failure have been reduced so far as possible There need be no hesitation in giving full dosage provided that it does not induce vomiting and the rate is not reduced below 80 or 90 beats per minute While continuing under digitalis, a ten days course of iodine is begun and the case is treated surgically when this is complete

It is becoming clear that few cases, whether they present auricular fibrillation or not, and with or without congestion of the venous system are unsuitable for treatment along these lines Although the risk from the operation is raised by the presence of failure, this risk is more than compensated by the fact that the chances of really substantial recovery, or of saving the patient from terminal cardiac failure, are very greatly increased by surgical intervention The operation should be undertaken even when after medical treatment *there are residual signs of failure Improvement of the circulation in* these cases is usually remarkable after operation

It is not unusual for the auricular fibrillation previously present to stop spontaneously within a few weeks of successful operation If it does not do so within three months, quinidine can be employed to arrest it if, on grounds previously considered (page 101), this is thought desirable

Thiouracil —This new remedy, a substance interfering with the endocrine function of the thyroid gland shows great promise The drug is given in doses of 3 grains (0 2 g) thrice daily Within two or three weeks the basal metabolic rate falls close to normal the symptoms largely subsiding exophthalmos and goitre, however, are little affected This improved state should then be maintained by the minimal dose required, usually not exceeding ½ to 3 grains (0 05 to 0 2 g) daily The treatment should be given only under close and experienced control Occasional adverse effects are conjunctivitis, urticaria, fever, enlargement of the thyroid gland, and decline in the number of polymorphonuclear cells Although rare the occasional occurrence of agranulocytosis on continued full dosage makes *a regular examination of the* blood imperative

This medicinal treatment will probably prove at least the best preliminary to surgical intervention, very possibly it may largely replace the latter, but it must not be used incautiously.

CHAPTER XXVIII

CONGENITAL MALFORMATION

Among the great variety of congenital malformations of the heart that occur, the physical signs of only a few are distinct. The chief pictures of clinical importance are described briefly in the present chapter. A number of the grosser defects are incompatible with extrauterine life or are survived only for a short period after birth. Different forms of congenital disease are particularly difficult to distinguish in very young children.

Persistent Ductus Arteriosus

This duct unites the arch of the aorta beyond the origin of the three main vessels to the left branch of the pulmonary artery. In the foetus it carries blood from pulmonary artery to aorta. The duct closes at birth, when the pulmonary falls below the aortic pressure, and it soon becomes impermeable and atrophies. In cases of its persistence the blood flows through it continuously from aorta into pulmonary artery. The characteristic sign is therefore a murmur waxing in intensity as the aortic pressure rises in systole and continuing in diastole until the next systole, this murmur is usually harsh, loud, and accompanied by a thrill. Its maximal intensity is at the 2nd or 3rd left cartilage, but being intense it is heard over a wide area at the base of the heart, and usually in the interscapular region, but not in the neck. Sometimes the murmur is cut short in diastole. When this murmur is heard, the diagnosis is almost certain. Continuous murmurs of an identical kind, however, are heard over anastomoses between large arteries and veins, these are usually in the limbs or neck, but occur occasionally within the chest as a result of bullet wounds. A continuous murmur varying in intensity with respiration and heart beat is heard over the jugular veins in some cases of severe anaemia. Although this noise is sometimes audible a little way down into the chest, it is unlikely to

be confused with a ductus murmur If in patency of the ductus the leak is sufficiently free, arterial signs similar to those in aortic regurgitation may appear, the pulse rising more steeply, the arteries throbbing more, and the diastolic pressure being lower than usual These signs are rarely more than slightly developed.

The defect causes no appreciable enlargement of the heart unless reflux is free It is often associated with dilatation of the pulmonary artery, which may give dulness in the 2nd and 3rd left interspaces near the sternum

It may occur alone or in combination with other malformations such as pulmonary stenosis Occurring alone and in its usual form, [illegible]

Ligation of the uninfected ductus is being advocated as a prophylactic measure, it is justifiable in the hands of the experienced, and is to be advocated in the infected

Bicuspid Aortic Valve

This is a very common defect, being found about once in every 100 or 200 autopsies It is not diagnosable, its importance lies in the frequence with which it is responsible for subacute bacterial endocarditis (page 186), about one fifth of the cases succumb to this malady.

Coarctation of the Aorta (Adult Type)

When the ductus arteriosus shrinks, the aorta sometimes becomes constricted at the insertion of the ductus, the obstruction is partial or more usually complete, the aorta appearing as though a cord were tied around it The constriction occurs just beyond the origin of the subclavian artery In complete or great obstruction the blood is carried to the aorta beyond the obstruction by greatly dilated anastomotic vessels, branches of the subclavian artery, especially scapular and internal mammary arteries, conveying blood to the lower aorta through the intercostal and superficial epigastric arteries, the defect is commoner than has been thought, it usually escapes diagnosis Sometimes the first sign found is the pulsation of [illegible] anastomotic vessels, enlarged and tortuous beneath the [illegible] vessel in examining the chest More often the anastomotic vessels

are found only after deliberate search has been made for them Hi[g]
blood pressure (200 mm Hg) is the rule in the brachial vessels, a[nd]
these and the carotid arteries often pulsate with unusual vigour, t[he]
main arteries at the base of the neck are often dilated, a systo[lic]
murmur at the aortic cartilage is frequent The post manubr[ial]
dulness of aortic dilatation may be found, and X ray examinati[on]
may show dilatation of the ascending aorta or the main branch[es]
The arch of the aorta cannot be traced in the radiograms It is t[he]
rule to see sinuous erosions of the lower borders of the ribs in anter[ior]
posterior X ray pictures, and this sign when conspicuous is di[agnostic]
gnostic, the erosions are caused by tortuous and dilated intercost[al]
arteries Whenever high blood pressure is discovered, the femor[al]
pulse should be examined, in cases of coarctation this is absent, [or]
it is feeble and occurs after the pulse at the wrist Signs of ventric[u]
lar enlargement are found clinically or by orthodiagraph in abo[ut]
half the cases The condition is quite compatible with a long life [and]
arduous work The average life is about thirty years Most of t[he]
patients are without material symptoms, the heart carrying on i[ts]
functions well for many years, the patients succumb to a variety [of]
maladies Cardiac failure and sudden death, usually from rupture [of]
the aorta are almost equally the most frequent causes, and accou[nt]
for about half the cases, others die of cerebral haemorrhag[e]
bacterial endocarditis renal disease, and of other and diver[s]
causes

Defective Interauricular Septum

Persistent foramen ovale occurs in 25 per cent of all hearts and [is]
not often of consequence The aperture may be large where mitr[al]
stenosis has developed

A malformation of the upper septum provides a rarer and wid[er]
opening, it is often associated with a small aorta The right heart a[nd]
pulmonary artery may become greatly enlarged The patient m[ay]
continue without symptoms in some, cyanosis and clubbing of t[he]
fingers persist for years X rays show a large globular heart, [a]
prominent conus, very large pulmonary arteries and a small aor[tic]
knob The E C G shows right axis deviation and often inversion [of]
T in leads *II* and *III* Death occurs in the average at 35 years a[nd]
may be from heart failure

A defective septum has no characteristic murmur, though the 2[nd]
pulmonary sound may be very loud Clots liberated from the sy[s]
temic veins are sometimes carried through it into the left hea[rt]
(crossed embolism) Auricular fibrillation may occur in the old[er]

cases and in those acquiring rheumatic fever

Persistence of a lower septal opening (ostium primum) is usually associated with defect of the mitral valve and mongolian idiocy.

Defective Interventricular Septum

The septum of the ventricles may be open in its upper membranous part, without other defect in the heart When the aperture is a full one the right ventricle is increased in size, and the X ray silhouette tends to be circular in outline A loud and long systolic murmur, *often accompanied by thrill*, and presumably caused by blood passing from left to right ventricle, is maximal about the 4th left rib near the sternum The murmur is usually well heard over the precordium, but is not conducted above the base of the heart or into the left axilla Cyanosis is unusual and there are few symptoms

The septal defect may be large and involve in greater or less degree the muscular partition, there may be no septum Admixture of the right (venous) and left (arterial) blood happens according to the extent of the defect The opening is usually associated with other defects such as malposition or transposition of the great arteries or with pulmonary stenosis or with suppression of the pulmonary artery *In such the work of the right ventricle is greatly increased*, and this chamber is of the size and thickness of the left In many the admixture of venous and arterial blood is free, septal defects are in fact the only common cause of cyanosis in congenital heart disease, the mixed blood being expelled into the aorta The resultant cyanosis is of the kind previously described as arising centrally (page 47), it may be transient and is then often slight, or it may be persistent and deep In the last case clubbing of the finger tips, and polycythaemia up to eight or ten million red cells, or exceptionally more, are usual, breathlessness is conspicuous and bodily growth is defective When clubbed fingers are fully developed, they are bulbous at their tips and the fingers are conspicuously cyanotic In these cyanosed cases too, epistaxis, haemoptysis, or cerebral haemorrhage may occur, and sometimes alarming cerebral symptoms appear in the form of attacks of an epileptic nature or of coma

In cases of septal defect, deep and persistent cyanosis, breathlessness and enlargement of the heart are the chief adverse signs, and yet some of the cyanotic cases live on into their forties Bacterial endocarditis, pulmonary tuberculosis, and other infections often terminate life, some of the cases die of acute heart failure

UNEXPANDED INFUNDIBULUM (PULMONARY STENOSIS)

Among congenital malformations, lack of full expansion of the infundibulum, the half of the right ventricle that forms the outlet to the pulmonary artery, is one of the most frequent When the defect is slight there is a little constriction between right ventricle proper and infundibulum, the moderator band forming one side of the constriction When less expanded, the infundibulum forms a small separate chamber, united to the ventricle by a narrow orifice below and to the pulmonary artery above The pulmonary valve is usually anomalous, being bicuspid or having its cusps actually fused together In general the less expanded the infundibulum the smaller is the stenosed aperture through which the blood has to pass out of the right ventricle, the nearer is the stenosed aperture to the pulmonary valve, and the more apt it is to be combined with a patency of the interventricular septum in these much malformed cases the aorta is often displaced so that it arises over the perforate septum (Fallot type) Unexpanded infundibulum with perforate septum is the commonest malformation yielding cyanosis In high grade stenosis much blood may be transferred from the right to the left ventricle and deep and persistent cyanosis appears with its accompaniments finger clubbing polycythaemia and breathlessness In high grade stenosis also, the right ventricle hypertrophies greatly and gives corresponding physical signs, including the E C G of right axis deviation

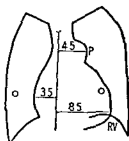


FIG 45 —Orthodiagram Child aged 12 weight 71 lb Probably congenital pulmonary stenosis Systolic thrill third left space attacks of cyanosis The patient has been rotated bringing the left shoulder forward a little The bulge of the greatly dilated pulmonary artery (P) is shown and below it an increased conus The apex of the heart is probably formed by the right ventricle

The stenosis displays itself through a harsh systolic murmur and thrill maximal to the left of the sternum, at the 2nd 3rd or 4th costal cartilage, but heard over a wide area of the upper chest as high as the clavicles it is transmitted to the interscapular region but not into the neck, very occasionally an early diastolic murmur is heard to the left of the sternum, and is caused presumably by pulmonary regurgitation The prognosis is variable a few patients living beyond thirty or forty, but the death rate is much above normal and especially in those much cyanosed and presenting much cardiac enlargement Infections often terminate life Despite the greatly

increased work of the right ventricle, failure with congestion is infrequent

Pulmonary stenosis of simple form is a very rare condition in which a strong membranous partition, perforated centrally by a small circular aperture, is found at the origin of the pulmonary artery.

Diagnosis

The diagnosis of congenital malformation of the heart should not be made upon the basis of a systolic murmur in the pulmonary area Such murmurs are among the commonest heard over the heart, they may be soft or harsh, and may be heard over the 2nd 3rd, or 4th left cartilages. In the great majority of instances the murmur is without significance, but may be used as a reminder that the base of the heart should be felt for thrills A diagnosis should not be made, though malformation is often rightly suspected, because a loud murmur *of unusual distribution is found over the* heart It is clear that the only sign may be a systolic murmur, but in such cases no great harm is done if the underlying condition remains undiagnosed The presence of a basal thrill, especially if this is maximal to the left of the sternum, is distinctly helpful Cyanosis, or a history of cyanosis, in cases of the kind, occurring on and off since birth, usually decides the diagnosis *Deep cyanosis that is* proved to be of central origin, and that is known not to be caused by the presence of an abnormal blood pigment, is almost diagnostic in itself, provided there is no venous engorgement and no disease of the respiratory system Otherwise the recognition of congenital malformation depends upon the presence of one of the characteristic groups of signs that have been described earlier

Prognosis and Treatment

It should be clear from what has been said already that the prognosis in cases of malformation of the heart regarded generally is by *no means so grave* as it has often been thought There are certain conditions which make extrauterine life impossible, or so precarious, [illegible] for many weeks or months but for the rest,

cyanosis and breathlessness, and enlargement of the heart and its degree

The malformations are not open to curative treatment The treatment is for symptoms as these arise The patient's activities are to be guided by the simple but sound principle that he lives within the limits of his exercise tolerance.

CHAPTER XXIX

CHILD BEARING ANAESTHETICS AND OPERATIONS

CHILD BEARING

HEART disease among young women is almost always rheumatic in origin Questions relating to pregnancy apply almost exclusively, therefore, to this form of heart disease, and especially to cases presenting mitral stenosis

RISK OF CHILD BEARING

Advice is often sought by young women with heart disease as to the risk they will run by bearing a child Often, too, advice should be given unasked to married women with heart disease Child-bearing entails an increased load upon the heart because the circulation has to serve both mother and child, and the mother in moving must carry extra weight During normal labour, a relatively heavy load is thrown upon the heart intermittently for a period of many hours In late pregnancy the heart is displaced upwards, and its action is impeded in unknown but probably not high degree The extra work involved by carrying is negligible in the first and almost so in the later months of pregnancy, the extra weight of the child is but a small fraction of the whole weight of the body The work done in labour varies, it is greater in primipara, it is rarely great enough in normal subjects to produce more than transient breathlessness and pulse-quickening, it is largely controllable by the manage- [illegible]

the risk proves in fact to be slight, for most married women of this class pass through one or more pregnancies without trouble There is, even in these, however, an element of uncertainty Judgment is being given, not as to advisability of undertaking certain acts at the moment, but of undertaking them in nine or more months'

time, and the patient's condition may not then be what it now is Thus the possibility of a renewed rheumatic infection needs to be considered on the basis of the past history, and a patient suspected to have suffered infection or reinfection recently should be advised to postpone decision for a year or more Cases with signs of enlargement of the heart and poorer exercise tolerance should always be advised that it is unwise to become pregnant The urgency of desire for a child will sometimes impel women to accept a risk that would not otherwise be taken the risk being realised, it is for their decision Women presenting auricular fibrillation run more risk during pregnancy than those without, the muscle of these hearts is in general less dependable, and work increases the rate of the beating unusually Those with fibrillation, those with large hearts and poor tolerance of exercise, and those with a past history of congestion should be told that the risk is much too grave, and every effort made to [illegible]

first

the

second

FAILURE AND CHILD BEARING

Because most women with rheumatic heart disease fail to seek advice, or, receiving it, are unable or unwilling to follow it a number of those who have serious cardiac conditions give birth to children The adverse effects upon the heart, and the increased death rate consequent upon child bearing tend perhaps to be exaggerated and for two reasons Firstly, heart failure will develop in many rheumatic heart cases during the usual child bearing age whether children are borne or not, secondly, many women attribute ill health to child bearing when they have no good reason for doing so Nevertheless, there is no doubt that the mortality among heart patients who marry is unusually heavy, and the effects of given pregnancies upon the subsequent condition are often obvious, or life is clearly brought to an end by a pregnancy Patients who develop congestion in the early months would almost certainly have developed the same degree of failure without becoming pregnant Failure develops chiefly in the later months In cases previously presenting normal rhythm or auricular fibrillation the onset may be gradual, with increasing breathlessness Or the onset may occur abruptly with the appearance of a paroxysm of tachycardia or of auricular fibrillation, these cases are often called instances of acute dilatation

of the heart When congestion comes it tends continually to increase and, if it becomes severe, the patient dies during the last few weeks of pregnancy or, more often, in the puerperal period Death may result from progressive failure, pulmonary oedema embolism, or infective endocarditis If the patient convalesces from labour, con gestion usually disappears quickly, a clear indication that pregnancy has been responsible Such cases may return to a condition seem ingly no worse than that preceding pregnancy, but often they are more breathless than previously, and among those who return to work congestion soon re develops

MANAGEMENT OF PREGNANCY

When a woman with heart disease takes to carrying a child she should cease to do some at least of her work Thus in the later months, all being well with her, she should cease to do her house work, to take any form of brisk exercise, or to undertake serious physical efforts of any kind She should spend hours resting each day, increasing their number as her time approaches The amount of exercise and the amount of rest taken will be controlled by symptoms, she must live without any distress of breathing

The appearance of venous congestion with or without auricular fibrillation in the first three months of pregnancy is an adequate reason for emptying the uterus, as soon as failure has been treated

Congestion is more apt to display itself in the later months, though it is then much more difficult to recognise in its early stages Oedema of the legs is of little value as a sign, a congested liver is displaced upwards by the uterus, and its edge and lower percussion border may remain indistinct Diagnosis then depends chiefly upon breathlessness, upon colour, and upon the condition of the veins The most important of these three is breathlessness at rest persisting during sleep Cyanosis is also a valuable sign, if general or if seen in skin that is warm, so are persistent râles at the bases of the lungs In examining the veins an allowance must be made for the increased pressure in the abdomen, normally the veins of the neck are dis tended more and to a higher level in pregnant than in non pregnant women Patients should usually come under strict nursing care *before actual signs* of venous engorgement appear Auricular fibrilla tion is to be treated with digitalis in the ordinary way, without any restriction of dosage The patients who react well may be allowed to go to full term, seemingly, natural labour is less harmful than induction Every care should be used to make labour as effortless

for the woman as possible An alternative is Caesarian section, now a good deal practised in these cases The cases are first fully treated for cardiac failure In those reacting well, the section is undertaken as soon as there is a good prospect of the child surviving and can be carried through in most cases with complete success In those failing to react well, delivery should be as soon as possible An advantage of Caesarian section is that it saves the mother from the last and most dangerous weeks of pregnancy The operation is often combined with a procedure that sterilises to rid the woman from further risk of pregnancy The justification or otherwise of this procedure is as much an ethical as a medical question

Patients presenting signs of venous congestion should not suckle their babies, but there is no reason to terminate lactation in simple valve cases

Anaesthetics and Operations

The dangers involved in anaesthetising and in operating upon

performed successfully, confidence grows and operations of less immediate urgency are undertaken Experience has widened greatly, and the results of very many hundreds of operations on patients suffering from heart disease have been recorded The actual risks are now known for all the chief forms of heart disease If we include all fatalities that can be connected even remotely with the heart, such as any case of pneumonia following operation, then the mortality attributable to the cardiac condition is about 5 per cent, this statement applies to unselected cases of conspicuous heart affections, such as mitral stenosis, aortic disease, auricular fibrillation and including all more serious maladies, it does not include trivial or doubtful cardiac maladies, it applies to groups of patients con
t

h
that prohibits operations being undertaken to meet a grave emergency, and that there is none that forbids trivial operations including tooth extraction, when these are necessary, under local anaesthesia

The advisability or otherwise of anaesthetising and operating upon cardiac patients is decided by a close consideration of the

disease from which the patient is suffering to determine actual risk This risk is then balanced against the benefits to be expected from operative interference The presence of cardiac disease increases the risk of operations, but in some cases the increase of risk is very slight and in other cases it is great

In the case of uncomplicated mitral stenosis or aortic regurgitation, or in that of any other subject able to follow his ordinary daily routine, the extra risk introduced by the cardiac state is practically negligible in contemplating surgical interference In cases of the kind, therefore, operations such as gastroenterostomy or the radical cure of hernia may be undertaken under general anaesthesia without hesitation from the standpoint of the heart

Unselected cases of auricular fibrillation, most of which are complicated by mitral stenosis and by enlargement, present an unexpected mortality of only 2 per cent, and here mortality can be reduced if operations of pure convenience are debarred in the more serious of the cases One of the most frequent operations performed in this group is that of thyroidectomy, the mortality is small Naturally all cases of fibrillation should undergo preliminary and thorough medical treatment with digitalis before operations other than urgent ones are performed The case of fibrillation and of flutter are almost the only ones to which digitalis should be given before operation, atropine should not as a rule be given to either

Paroxysmal tachycardia does not contraindicate operations under anaesthesia, though the onset of a paroxysm would naturally postpone any that was not extremely urgent

In hypertension and in breathless cases of arteriosclerosis the ordinary risks of anaesthesia and operation are raised about 5 per cent The subjects are elderly, and post operative broncho pneumonia, which is the chief cause of death, is a little commoner than in more normal subjects of the same ages Prostatectomy is a frequent operation in this group and is well borne as a rule Patients presenting definite evidences of renal insufficiency show a high mortality

The presence of mild angina pectoris does not contraindicate general anaesthesia, but should rule out operations of pure convenience and these only We come to cases running greater risks

In severe angina, syphilitic aortitis, and aneurysm, operations that are not urgent are rarely advisable, for among these patients the added mortality is 10 to 20 per cent In severe angina chloroform has sometimes been given to stop pain, and with impunity

In no case of coronary thrombosis, unless there has been good

recovery and many months have elapsed since the event, should operations other than those necessary to save life be performed

Cases of failure with definite venous congestion will sometimes require surgical treatment in emergency Otherwise the cases should be treated thoroughly and as bed cases before operations are considered, and the treatment should be prolonged for weeks or months after signs of venous congestion have disappeared Operations definitely calculated to improve general health may then be undertaken, but not operations of convenience It is particularly to be noted that elderly patients with venous congestion are not good subjects

Heart cases stand general anaesthetics well, ether is well tolerated by the young and middle aged, but should be avoided in older subjects that are bronchitic There is no special danger from the use of chloroform in heart cases Because this and other general anaesthetics are well borne, spinal anaesthesia is rarely necessary If gas and oxygen are used, asphyxial signs must be avoided quite strictly There is no objection to carbon dioxide in amounts sufficient perceptibly to deepen breathing A more important consideration than the kind of anaesthetic is the competence of the anaesthetist Quick induction, to avoid struggling, is essential, speedy emergence from anaesthesia is desirable

In heart cases of any kind during natural labour, chloroform is still the anaesthetic to be preferred, though gas and oxygen may be used General anaesthesia is advised for Caesarian section

It is important to realise how the fatalities occur Neither anaesthesia nor operation imposes, so far as I am aware, any burden or stress upon the heart When cardiac patients submit to operation under general anaesthesia, those that die unexpectedly do not usually succumb under anaesthesia, but in the hours or days that follow The reason for many of the deaths is that patients who are suffering from the graver forms of heart disease are relatively intolerant of infections and exhaustion Thus post operative infections, such as *broncho pneumonia*, the commonest cause of death in elderly heart cases, or a large wound area left infected after operation, are not well borne, neither is the intoxication that sometimes *follows acutely upon thyroidectomy*, these complications are ill tolerated because they raise the heart rate while rendering the muscle less efficient for its work Coronary thrombosis and cerebral thrombosis are *not infrequent after events in elderly subjects*, they cause fatalities, which are associated perhaps with feebleness

of circulation In mitral stenosis with or without auricular fibrillation, the fatal event may be embolic, and in these and high tension cases pulmonary oedema may supervene Patients who present failure with congestion, or are threatened by it, before operation, may progress to fatal venous stasis in the few days that follow

Sudden catastrophes during anaesthesia or operation occur, but are unusual Much of what is said and thought about fatalities in cardiac disease under anaesthesia is due to a very widespread misconception It is customary to attribute a large proportion of deaths occurring under anaesthetics to acute failure of the heart There is good reason for doing so Thus it has been shown that under light chloroform anaesthesia fibrillation of the ventricles is the usual cause of death It is quite erroneous to suppose that the fault lies in these cases in the heart, it lies either in the method of administering or in the unfortunate choice of anaesthetic This accident is particularly frequent in the case of children, it is a phenomenon displayed by perfectly healthy hearts There is no evidence to suggest that any reduction of such fatalities will result by excluding from anaesthesia patients in whom a systolic murmur, extrasystolic irregularity, or other isolated sign attributable to the heart has been detected And the exclusion of cases presenting serious heart lesions could not reduce this mortality appreciably, for it is clear that only a very small proportion of patients dying under anaesthetics are properly to be regarded as heart cases A real understanding of the practical problems will not be reached by enquiring what proportion of deaths under anaesthetics and operations can conceivably be attributed to the heart, but by enquiring what is the ascertainable risk in cases presenting clear evidences of heart disease That there is an increased risk from anaesthesia when the heart is recognisably unsound is clear enough but, where anaesthetic deaths in general are concerned, this becomes a trivial issue It is an important issue when it concerns the individual heart case requiring anaesthesia for operation, and it is from this standpoint that the problem has been discussed in this chapter.

CHAPTER XXX

DIAGNOSIS, PROGNOSIS, AND TREATMENT

Diagnostic Terms and Summaries

In medicine generally diagnostic terms are often indispensable They have particular value when they name specific diseases running well defined courses and calling for definite practical measures The name "diphtheria" at once suggests isolation and specific treatment "Cancer of the stomach" is a name that not only conveys a well defined clinical picture but a disease running to a fatal termination

The satisfactory use of short diagnostic terms in cardiac disease is very difficult The system of names for cardiac disease in the last century was mainly anatomical it was this system which gave such undesirable emphasis to defects of the valves, the harmful effect of which, though now lessening, is still with us These names of valve affections are quite inadequate To speak of "aortic dis ease" conveys neither the origin of the malady, nor the prospect of life, nor the need of treatment, the malady may have arisen as part of rheumatic carditis or of syphilitic aortitis, the man may be moribund or in almost perfect health and requiring no treatment In this old nomenclature there were few or no names that had the striking and helpful significance of those previously mentioned We now possess a number, for example, "subacute bacterial endo carditis", "rheumatic carditis", "syphilitic aortitis", "auricular fibrillation", "auricular flutter", "coronary thrombosis", "hypertensive failure" Each of these frequently fills a definite and most useful purpose This list might be lengthened with advantage The terms "aortic disease", "mitral stenosis", in speaking of cases, might profitably be retained and confined to those instances that are almost uncomplicated, cases up and about their business in life

Cases that have general venous engorgement of cardiac origin should be called "failure with congestion" or, shortly, "congestion", irrespective of other cardiac findings The term "cardiac asthma"

may be employed for cases of failure presenting nocturnal attacks prominently Short terms of the kind we must have for everyday convenience A term to be serviceable must emphasise the main feature of the case and this may be a mechanical defect, a bacterial invasion, a degenerative process, a disorder of function it is a diverse nomenclature No system of diagnostic terms has yet been devised which really fills this need of briefly summarising cardiac cases, this is so because the many structural defects, and the very many dis orders of function that occur, combine in an endless variety of ways For the same reason the systematic classification of heart diseases under names is scarcely possible It would seem that the attempt to find an inclusive series of terms must be abandoned Such brief terms as those that have been named will continue to be used, owing to their convenience in everyday conversation The further practical need is of a simple system whereby we can place on brief record all the essential features of a case that are required for prognosis or for treatment, so that these may be gathered at a glance Such a system I have now long used and found adequate to its purpose The summary of the case is recorded in eight lines

	a	b	c	d	e
1 Exercise tolerance	None	Poor angina	Good	Poor	Poor
2 Congestion	+				
3 Enlargement	Mod	Def		Consid	Mod
4 Rhythm	A F	P A			
5 Valves .	M S		Syst M	A R free	A R sl
6 Infection .					Subacute b
7 Vessels .		B P 200		Aneur	
8 Etiology .	Rh			S	

The table here given provides a synopsis of five patients

a Breathless at rest, congestion, moderate enlargement of heart, auricular fibrillation, mitral stenosis, rheumatic origin

b Exercise tolerance poor through angina, slight definite enlarge ment, pulsus alternans, hypertension

c *Good exercise* tolerance, systolic murmur in mitral area

d Poor tolerance considerable enlargement, free aortic regurgita tion, aneurysm, syphilitic

e Poor tolerance, moderate enlargement, slight aortic regurgita tion, subacute bacterial endocarditis

The short dotted lines indicate that in the corresponding spaces

there is nothing significant to record The degrees of enlargement here referred to are defined on page 127

Prognosis

A NORMAL HEART

If after carefully examining a patient and finding nothing definitely wrong we are to fear that vital things are still undiscovered, we shall remain at the mercy of every rumour having patient or patient's heart as its source, and be incapable of responsible decisions [illegible]

and o [illegible]

healtl [illegible]

rest on knowledge and experience, it is not to be substituted by [illegible]

soundness is its power to engage fully in its work without complaint No patient having normal capacity for bodily exercise has grave [illegible]

blood supply proving inadequate, cause anginal pain Thus the presence of good exercise tolerance can be relied upon to exclude all the more serious maladies, though it should be understood that full capacity for work cannot be gauged always and entirely from the patient's own statements, valuable as these usually are Actual experience will show that only exceptional patients, having such full capacity, will present clear signs of disease

Because there are these exceptions, full tolerance of exercise is an [illegible]

blood pressure, though the last two are so very rarely accompanied by normal exercise tolerance that it is hardly necessary to name them A short physical examination suffices to exclude the states of disease named particular heed being given to the valves, for it is this form of disease which is most compatible with normal tolerance The heart must be listened to while the patient stands lies, and lies after exercise on the left side Much enlargement of the

heart and progressive mischief, syphilitic or rheumatic, are incompatible with good exercise tolerance, though the physical examination is an extra safeguard There must be a brief enquiry for paroxysmal disorders of heart action and, these also excluded, the heart may then be proclaimed sound A systolic murmur over any part of the heart should lead to a sharper search for more definite evidences of disease and, in young people, to a close enquiry for recent rheumatism Neither the *systolic murmur, nor a modification of a heart* sound, nor a sound interpreted as exocardial, nor an unusual form of electrocardiogram should deter us from pronouncing the heart essentially sound in these circumstances In this connection it should be noted that an isolated sign rarely has much significance Indications of disease are grouped together almost always The foregoing are the criteria that were used on a very large scale in judging men fit for active service during the 1914 war they proved themselves to be reliable

A more difficult case is where exercise tolerance is reduced in the absence of physical signs of disease in heart, lungs, or other organ, for very many such patients have normal hearts Here for the time being we must proceed by rule of convenience, declaring the heart sound in the young but holding it under suspicion or to blame in older subjects (see page 174).

CHRONIC HEART DISEASE AND PROGNOSTIC GROUPINGS

In considering on what general lines an opinion of the expectation of life is to be formed in a case of chronic heart disease, we may profitably ask whither we are led by subdividing patients systematically into groups All cardiac patients may be divided up into

the heart, unenlarged or enlarged in one of several degrees They could be subdivided again and again according to the nature of the valve defect and its degree, according to etiology, to blood pressure,

practised in this way, the finer the subdivision the more cumbersome and impractical it becomes It is imperative to proceed upon a broader basis, and to emphasise those features of cardiac disease that are of chief prognostic significance

Stated in the order of their importance, and omitting active in

fection for purposes of simplicity, the following factors mainly govern prognosis

(a) The state of the circulation at rest, or under exercise as judged by breathlessness, and the presence or absence of anginal pain in similar circumstances These considerations are essential and intimate parts of any sound prognostic system, they are so important that prognosis of chronic disease carried through purely on this basis would not prove very unsatisfactory

(b) The size of the heart

(c) The presence or absence of syphilitic disease of the aorta

(d) The presence or absence of gross disturbances of heart rhythm (for example, auricular fibrillation), the presence or absence of high tension

(e) The state of the valves

Before proceeding to deal with the practical problem of prognosis from this standpoint, we must first understand quite clearly what is required No enquiry, however minute, would carry our prophetic powers to such a pitch that we could state definitely how long a patient has to live It is not asked We are asked to give an opinion along the following simple lines

1 Does the expectation of life seem normal?

2 Has the patient many years to live?

3 Has he a number of years, say ten, a little more, or less?

4 Has he but a few years?

5 Is life to be measured in months, a precarious life of perhaps one, of perhaps two years?

6 Is the patient in imminent danger or actually moribund?

These questions can usually be answered accurately The first has been answered in enquiring how a normal heart is recognised The remaining questions may be answered in tabular form Sufficient types are here included to illustrate fully, the attempt is not made to cover all possibilities It is assumed that the cases are all under full and proper treatment and that none are suffering from active rheumatism, or from infective endocarditis

1 *Many years' expectation*

Valve disease in young adults, with little or no sign of cardiac enlargement, no auricular fibrillation, and good or fair exercise tolerance

2 *A number of years' expectation (about ten years)*
Angina of effort, early cases
High tension, little or no sign of enlargement, fair tolerance
Auricular fibrillation with or without valve disease, but little enlargement and fair tolerance
Enlargement moderate, with or without valve disease, but with fair tolerance, and normal rhythm

3 *A few years' expectation (about three to six years)*
Angina of effort on walking a short way (with or without previous coronary thrombosis)
Syphilitic aortitis
High tension with moderate enlargement or angina
Enlargement great or moderate with poor tolerance
Auricular fibrillation and moderate enlargement
Failure with congestion (relieved by treatment)

4 *Living precariously up to one or occasionally two years*
Enlargement great, congested
High tension and cardiac asthma
Congestion unrelieved or in syphilitic aortitis, in high tension, following angina, or after coronary thrombosis
Angina unrelieved by rest

5 *In imminent danger*

Oedema of lungs Coronary thrombosis (recent) *Heart block and syncope (epileptic state)*	*Often recovering*
Leaking aneurysm Anginal state Gross failure with congestion, unrelieved	Rarely or never recovering

It is not intended that this table shall form a precise guide to prognosis in individual cases, each of which must be considered upon its own merits as discussed in earlier chapters It is only intended that it shall broadly and clearly illustrate the relative importance of the chief prognostic factors previously named, and how these come to be used in answering the relatively simple questions that are asked It will be observed that, apart from the first group of cases, valve disease can be made to take little part That is so because, as we proceed to more and more serious categories, other factors become more and more decisive, and valve disease becomes relatively unimportant

CONVERSING WITH THE PATIENT AND HIS FRIENDS

To a sick man his doctor's visit is the chief event of the day Cardiac patients react quickly to the atmosphere surrounding them, they must be helped to keep or gather courage Cardiac cases are naturally sanguine, depression, when it comes, usually derives from others It is the doctor's plain duty to enter the sick room with cheerfulness, to give counsel thoughtfully but confidently, and to leave behind as he goes an appropriate word of encouragement, to comfort the patient in his waiting, to help his suffering, or to allay his fears A countenance of gloom is as out of place in a sick room as is a coffin

In speaking with a patient or his friend about the nature of the malady, truth of statement is a first essential It is not always expedient to volunteer the whole truth, but answers must be given to direct questions, evasions usually defeat their purpose When a man in his right mind demands to know if he is dying, as seldomly he does, he should have his answer, and, if it is definitely adverse, it can be conveyed compassionately Spontaneously to acquaint a patient that his life is in grave danger is rarely necessary or advisable It sometimes becomes desirable to convey to him, directly or through his family or legal adviser, that it would be a simple act of wisdom if he set his house in order

To patient or to patient's friend simple and unmistakable language should be used, veiled statements are too apt to be misinterpreted The words must be chosen thoughtfully The word "disease" should never be used, disease of the heart conveys at once to most patients the idea of something incurable and threatening The language should be as untechnical as it can be made A patient has a right to be told what ails him, if he so desires, in terms that convey an idea of the magnitude and significance of the trouble, he has no right to technical diagnostic terms, and it is very rarely wise to parade these before him Accurate information cannot be conveyed by means of strange words, which to unaccustomed ears bring unintended meanings, and for many the word once caught up becomes a matter requiring search in medical books, or a topic of debate with other patients Thus the word "angina" should never come first from a doctor, if it is understood at all it will convey in almost all instances an ominous meaning, yet the prognosis varies up to fifteen or more years The word "dropsy" is to be avoided, and the adjective "malignant" eliminated completely from the medical man's vocab

ulary The valves should not be named, nor murmurs mentioned These names and details should be of no concern to patients Methods of examination likewise require little or no explanation, a clear example of the unhappy effects of unnecessarily disclosing technical detail is the well known instance of high blood pressure readings

It is rarely desirable to speak of catastrophes that may arise out of over exertion, though a warning that certain acts are foolish and endanger life is at times necessary, but only in such cases as those of grave angina, syphilitic disease of the aorta, or failure with congestion Dramatic utterances are undesirable, and do not pass the lips of competent doctors The medical attendant who tells his patient that he or she "may drop dead at any moment", a not very unusual phrase, is guilty both of cruelty and folly Apart from its total lack of consideration, sudden death in heart disease is never so inevitable as to warrant such a statement In grave angina, and in thoracic aneurysm, sudden death is frequent, but in most other common cardiac maladies it is comparatively rare In the named instances, and in these only, if the question is directly asked by the patient, it must be answered that there is the chance, such information should not be volunteered to him though it is wise to tell a near and discreet relative that the life is a precarious one It is important that the motive should be correct, that the statement should be made chiefly out of solicitude for the patient's safety, or withheld out of consideration for the family's mental comfort A medical man who fails to warn relatives of a possible sudden ending may be a little blamed if and when the event occurs, but a doctor who sets out to safeguard himself against all risks of this kind, deliberately prefers his own to others' interests Death without warning disturbs a patient's family enough, *its long, and often unnecessary, anticipation inflicts more* suffering

A few well chosen words are far better than a diffuse statement out of which any detail may be seized upon and emphasised, while the real substance is forgotten It is most necessary to be brief and to keep to essentials

Every man will have his own way of saying things, it is not the precise form of expression that matters, it is the outlook A doctor's watchword should be optimism and not pessimism, he should be hopeful, and not fearful of the unknown, and experience will show that this profits both his patients and himself A statement of prognosis will be formed on the lines laid down elsewhere, and if the

prospect is uncertain, then it is the brighter prospect that should, within reason, be presented to the patient

TREATMENT

The treatment of heart disease consists chiefly in managing cardiac failure and angina pectoris of different grades of severity The treatment of a patient convalescing from failure with congestion has been discussed at length, so has that of angina pectoris and of hypertension Special points of treatment have been considered in relation to the several conditions to which they apply Thus most of the ground has been covered There is still something that may be said usefully to ensure that the management of heart cases that are up and about should be understood There are supplementary statements, which, included here, save their reiteration in several places in this book

Healthy habits—Experience has led us to recognise healthy habits of living Very prudent people live quietly and moderately, they have their simple daily routine of work and pastime, enlivened by occasional excursions and entertainments, social gatherings and visits to and from their friends They are temperate in their eating taking no more than will maintain them in robust health, arranging their diet to consist chiefly of plain good food, relieved occasionally of its monotony by a more elaborate, but not heavy, meal They are regular in their habits of work and exercise, and in their meal times They are strictly temperate in their drinking They control their emotions and their passions They avoid all forms of excess They use tobacco little if at all They welcome the freshness of abundant air and open spaces, delighting in the feelings of invigoration that accompany active exercise, they love the warmth of sunlight playing on their skins, and the sleepiness of healthy fatigue These are habits that few people in industrial countries now adopt, that fewer still maintain The cares and distractions of an increasingly complex life, indoor or sedentary occupation, advancing years and decreasing energy, interfere less or more, and the prudent rules are neglected, neglect is the easier because the penalties, owing to the body's great power of resistance to disease, are uncertain in their incidence and often long deferred Healthy habits would become more prevalent if the consequences of their neglect were more immediate and invariable They are largely the habits of the young, to which many older people rapidly return of their own accord in

convalescing from some acute but passing illness While immunity from consequences is often long enjoyed by the originally healthy, it is not enjoyed to the same degree by defectives Thus a regular life, which may be said to mean unusual prudence in the former, is a necessary safeguard to the latter, and among these we include all patients suffering from well defined disease of the heart

Avoidance of infection —For cardiac patients infection is a special danger Reasonable precautions will not eliminate infection but will reduce the frequency of its recurrence Crowded rooms and gatherings in public buildings should be avoided, especially in the winter season, and at such times as infection is known to be prevalent Those who are infective in the same house should keep their distance, and visitors in an infective state should be discouraged by the household A further safeguard is a scrupulous hygiene of the mouth and throat

Tobacco —It were best that cardiac patients did not smoke at all, but if they will it must be done in very strict moderation We may not know definitely that long continued smoking is injurious to the blood vessels but we have reason strongly to suspect it It is clear enough, however, that smoking is responsible, in a very large number of its habitués for much strenuous coughing, that it leads in many people, by irritation of the respiratory passages, to infections of the throat and of the trachea Tracheal and bronchial irritation and infection are particularly prevalent among but are not confined to those who inhale the smoke, and in these a winter cold is often followed by a period of weeks or even months of morning cough Such coughs and infections are most injurious to many heart cases

Exercise and manual work —In all cardiac cases that are fit to walk about outdoor exercise is beneficial, for it encourages health of mind and body The principle to be laid down for these patients is that they live strictly within the limits set by their symptoms Patients who suffer from breathlessness may take exercise that makes them breathe a little rapidly from time to time, so that they begin to be conscious of the breathlessness, but there must be no pushing on to a point of distress, or to a point where the sense of breathlessness is not lost almost as soon as rest is taken So, too, with anginal pain, the man must live strictly within the limit of his pain, stopping at once when it is felt, and avoiding repetition of the act In neither one case nor the other must so much exercise be taken as subsequently to bring about unpleasant fatigue. It is useless to attempt

to lay down beforehand precisely what a man may or may not be permitted ultimately to do It is for each patient a matter of cautious trial, and, if a trial is carried too far, then a rest period of hours, or in some cases days, should follow as compensation Exactly the same principle applies to work, the work should suit the case and be gauged by trial Cardiac patients can rarely engage in heavy manual labour, such as farm labouring, heavy hammering, or heavy porterage, some are fit for moderate manual work such as bricklaying and carpentering, most will find lighter work, such as painting or light fitting or purely sedentary work, most suitable It is often better to curtail the hours than to attempt to change the work A patient should not be told to leave his job, unless there is quite clear reason for his doing so, and unless it is thought he will comply Occupation of mind and body banishes anxiety and cultivates contentment Children should continue schooling although bodily activities may need strict limitation, and in the young must be controlled closely

Patients who possess an indifferent tolerance of exercise should report frequently, declining tolerance is a matter for concern It is unsound to allow congestion gradually to develop without interference, a fall in respiratory reserve or suspicion of venous overfilling should at once suggest a short period of rest in bed Early treatment by rest is economical, for it often postpones attacks of serious congestion and the long periods of rest that these involve

Strenuous acts, such as running walking fast uphill, walking against strong winds, lifting weight in excess of 50 lb , struggling of any kind, should be forbidden for all time to patients that have once suffered from cardiac failure to anginal cases, syphilitic aortic disease, and to cases recovered from coronary thrombosis And the same patients must be instructed to avoid situations in which they may be tempted to perform such acts in emergency Thus no such patient should walk on broken or precipitous paths, or drive an automobile into the country unattended, they must look to others to be the first in helping in accidents or emergencies that are liable to strain the helper

Diet —The diets here given are intended only to guide and not to be used rigidly, quantity and form of food may be varied within reasonable limits to suit individual cases

DIET I

Grave Congestion, about 750 Calories

Breakfast (8 9 A M)
(a) Rusks 1 oz , butter (unsalted) ½ oz
and 1 cup of tea (with milk and 1 2 lumps of sugar) 5 oz (with or without syrup or ho
or (b) 1 raw egg in 5 oz of milk

Dinner (1 30 P M)
Minced fresh meat or chicken 2 oz or fresh fish 3 oz.
Boiled or mashed potato 2 oz , or macaroni 2 oz
Stewed apple or plum 2 oz.

Supper (7 P M)
(a) Custard 5 oz
or (b) Milk pudding 5 oz
or (c) Milk 4 oz , shredded wheat 1 oz

Drinks in small quantities between meals Total fluids including tea and milk in above dietary, rarely to exceed 20 oz (570 c c) This diet should contain not more than 10 grains (0.7 g) sodium chloride.

DIET II

Convalescence, without Exercise, 1000 to 1400 Calories

Breakfast (8 9 A M)
(a) Rusks 1½ oz
Butter (unsalted) ¼ oz
1 egg, poached or boiled, or fresh white fish (not fried) 3 oz.
Tea or coffee 4 oz
Milk 1 oz
Sugar 1 2 lumps
Honey 1 oz
or (b) Grapefruit or orange
Cereal, dry cornflakes or shredded wheat 1 oz or cooked oatmeal 3 oz
Milk 5 oz
Sugar ½ oz
Rusks 1 oz
Butter (unsalted) ¼ oz

inner (1 30 P M)
(a) Fresh white fish (not fried) 4 oz (or meat, chicken, rabbit, mutton, sweetbread or game 2 oz)
Potato (boiled or mashed) 3 oz
Tomato, green of cabbage, peas or beans or salad 2 3 oz
b) Milk pudding 5 oz
with cooked fruit (prunes, plums, apricots, baked apple) with sugar, or raw orange, peach or grapes

Tea (4 5 P M)
Tea 4 oz
Milk 1 oz
Sugar 1 2 lumps

Supper (7 P M), only one course
(a) Fish 4 oz
Potato 3 oz
(b) Milk pudding 6 oz
(c) Chicken 3 oz or fish 4 oz
Salad

oedema

Bread may be substituted for rusks when there is no oedema

All meals to be taken without fluids Fluids to be drunk an hour before chief meals, water or, for those accustomed to alcohol, whisky or brandy (½ oz) and water not more than twice daily, or once with 1 glass sherry, claret, hock with midday meal Total fluids up to 40 or 50 oz

Diet I is suited to grave failure with congestion with oedema, to acute coronary thrombosis, to grave angina pectoris at rest Such a diet can be maintained up to a week without causing serious loss of strength As a case of failure with congestion progresses to convalescence the diet is increased until it corresponds to Diet II

Diet II is equally suited to other cardiac cases such as angina pectoris, or auricular fibrillation under active treatment with digitalis that are confined to bed, or to cases of hypertension or senile heart sitting in their chairs Where convalescence is more advanced and bodily work is undertaken, more food will be needed

Decrease in the starch content of the diet may be required if flatulence is present Flatulence often results in undue breathlessness and disturbed rhythm and not infrequently determines anginal pain It is necessary to insist that no fluid is taken at meal times

For cases of chronic heart disease that are free from major symptoms there
be regular,
foods being
that, while well nourished, they should not become obese, obesity already present should be treated

This book has been supplemented for those who desire information regarding "Vascular Disorders of the Limbs" *by my more recent small book published under this title*

INDEX

THE END

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